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THE GENESIS OF THYROID PROTEIN*

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RESEARCH in endocrinology has taken a new viewpoint in recent years. A decade ago, interest still centered about the addition or subtraction of effective hormones from the animal or patient under observation. At present, however, interest concerns the regulation of the output of the animal's own hormone factories, and the conditions of hormone manufacture. It is no longer enough to administer hormone from an outside source to a hypoendocrine patient or to injure or remove the gland of the hyperendocrine patient. With the discovery by Uhlenhuth, Loeb, Collip and others of tropic hormones, we now seek to control the activity of natural hormone factories with specific chemical substances derived from the pituitary.

At present, however, the study of these various factors is in a very confused state. Not only do the various hormones antagonize each other—thyroid vs. pancreas, for example—but as shown by Hisaw,[1] tropic hormones vie one with another in stimulating or suppressing the respective glands that they regulate. Attention has thus been directed to the chemical processes by which hormones are manufactured and stored in the body, because the effects of the tropic hormones are primarily concerned with the biochemistry of the endocrine glands themselves.

From this angle the thyroid offers at present a most hopeful point of attack, both because the chemistry of its hormone has been thoroughly studied by Kendall[2] and by Harington,[3] and because its histologic response in various physiologic and pathologic conditions has been well correlated with these chemical phenomena. In the maze of contradictory evidence as to the modus operandi of various tropic hormones, a heartening note is the clear-cut fact that the output of the thyroid in a hyperplastic gland can be regulated under favorable circumstances by a simple and relatively nonspecific substance—elementary iodine.

It is the purpose of this paper to discuss the action of iodine in the hyperthyroid animal or patient from the standpoint of the chemical effect that it produces in the gland itself.

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THE RESPONSE TO IODINE IN HYPERTHYROIDISM

First of all, however, let us consider one or two salient features attending the effect of iodine in hyperthyroidism. The first of these is well illustrated by an observation that Segall[4] made upon himself at the Massachusetts General Hospital some years ago. Segall raised his basal metabolic rate to about plus 30 per cent and maintained it at this level by a continuous daily consumption of a rather large dose of thyroid substance. While continuing this daily dose, he took iodine in generous amounts in the form of Lugol's solution. No change in his basal metabolic rate ensued. He concluded that iodine does not relieve hyperthyroidism of exogenous origin, and this fact alone would strongly suggest that its effect, directly or indirectly, involves the animal's own thyroid.

A second feature of iodine therapy is the quantitative nature of the response to varying doses of iodine, as studied by W. O. Thompson and his collaborators.[5] The response can be described roughly as a mass-law phenomenon, in that, within certain limits, larger and larger doses produce smaller and smaller changes in metabolism. This effect must be borne in mind if we wish to explain the results of iodine therapy on a rational basis.

Finally, there is iodine tolerance, as studied by Perkin[6] and his collaborators. They found that, in the patient whose hormone factory is delivering large amounts of its product into the systemic circulation, there occurs a rapid removal of newly administered iodine from the blood stream.

These several phenomena all suggest, of course, that the administration of iodine in hyperthyroidism must produce a profound disturbance in the manufacture of hormone. They suggest, indeed, that the effect of iodine therapy is intimately related to the genesis of thyroid protein.

THE DEPOSITION OF NATURAL THYROGLOBULIN

The normal thyroid consists of many individual follicles arranged in groups that resemble bunches of grapes. Each follicle is apparently a functional unit. It is filled with an eosin-

staining, albuminous material, colloid, and is encased in a meshwork of capillaries, nerves and lymphatics. In human, colloid goiter, as obtained at operation, the follicles vary considerably in size. Many of them are cystic and overdistended with colloid. In contrast to these are the raisin-like, hyperplastic follicles of exophthalmic goiter, in which the characteristic eosin-staining colloid is replaced by a pale pinkish material, scant in amount. The most striking histologic effect resulting from the administration of iodine to a patient with exophthalmic goiter is the rapid filling up of such follicles with newly formed colloid. As a result of this phenomenon the clinician feels the gland becoming smaller and harder in the course of a few days. Coincidentally, the histologist finds many of the follicles have become spheroidal, and so filled with eosinophilic material that, at first sight, they might be mistaken for normal thyroid or colloid goiter tissue. At the same time the biochemist is able to demonstrate a rapid accumulation of iodine within the gland, which may increase the iodine content tenfold or more. Thus, both chemical and microscopic studies emphasize the appearance of newborn thyroid protein in a striking way.

Why does the administration of a surplus of iodine help the hyperthyroid patient? What is the mechanism that causes the hormone to accumulate in the gland, and, as Lunde[7] and Curtis[8] have shown, to be depleted in the circulating blood? Why should an apparent block in the delivery of hormone from the natural factory occur coincident with clinical improvement? The answer seems not to reside in a change of the activity of the follicular thyroid cells themselves. Marie Krogh, Lindberg and Okkels[9] have studied the abatement of hyperthyroidism induced in guinea pigs by thyrotropic (pituitary) hormone. Friedgood[10] in Boston has made similar observations. Both agree that the elaborated Golgi apparatus and other intracellular evidence of high secretory activity remain unchanged during the response to iodine. Although it is by no means clear that Graves' disease results from an excess of thyrotropic (pituitary) hormone, nevertheless, the changes in the thyroids of such patients when treated with iodine are nearly identical with those of treated animals. This fact warrants our considering the respective changes within the two types of gland as essentially the same phenomenon. Both types of gland retain their hyperactive cells at a period when new hormone is flowing into the follicles, and little is being released.

A clue to this problem was suggested by the work of Gutman, Benedict, Baxter and Palmer,[11] who studied the accumulating thyroid protein in glands of patients at various stages of iodine therapy. They found that the iodine first bound in the hormonal protein comprised little or none in the form of thyroxine. Subsequently, as more iodine accumulated in the gland, the relative thyroxine content increased even faster than did the total iodine. Eventually, however, the later increments of thyroid iodine (protein-bound) were laid down in a constant proportion, so that the new thyroid protein retained about one third of its iodine in the form of thyroxine. This protein, as we know from other experiments, is an excellent remedy for human myxedema. Thus one can observe in the course of iodine therapy not only the accumulation of iodine-containing protein, but also its transformation from an irregular to a normal hormone. Furthermore, it seems probable, from the following evidence, that this final step in the genesis of the thyroid hormone may be effected by a relatively simple chemical process in accordance with the mass law. To test this hypothesis we propose to demonstrate that such a transformation of a near-hormone into highly potent protein may be accomplished in vitro without the aid of vital activity—at least, without anything more vital than a crystalline proteolytic enzyme.

THE PRODUCTION OF ARTIFICIAL THYROID PROTEIN

It is evident from our previous discussion that, given elementary iodine and the ordinary constituents of protein, the organism is able to manufacture thyroglobulin at a rapid rate. Of the various factors involved in this complicated process, the action of enzymes is most important. In order to simulate the natural production of thyroid protein, it would be ideal to start with elementary iodine and simple amino acids. Although it is possible that this procedure may some day be achieved, it will require a technic that is, at present, beyond reach. It is true that Taylor[12] made a step in this direction when he produced a protamine from simple amino acids using enzymes as synthetic catalysts. For the present problem we propose to assume that large fragments of the thyroglobulin molecule are actually produced in body cells in some such manner, and that eventually the body cells (or thyroid cells) have available a long, peptide chain containing iodine in the form of diiodotyrosine or a simple derivative thereof. The last step in the synthesis in vivo is the most important one, because it produces an active material from an inert substance. It is to our best interest to simulate this last stage.

In the search for a starting material to produce artificial thyroglobulin, namely, an inert substance containing iodine in a long peptide chain, diiodotyrosine peptone was selected as the logical one. Fortunately, in splitting up natural thyroglobulin with enzymes, Harington and

Salter[13] had found that fragments of the molecule containing thyroxine precipitated out of solution at an acidity of pH 5.0, whereas the major portion of the peptone, containing diiodotyrosine, remained soluble. Thus, enzymic hydrolysis of thyroglobulin yields two peptone mixtures, which are separable, one, insoluble, consisting of thyroxine peptone and the other, soluble, consisting of diiodotyrosine peptone.

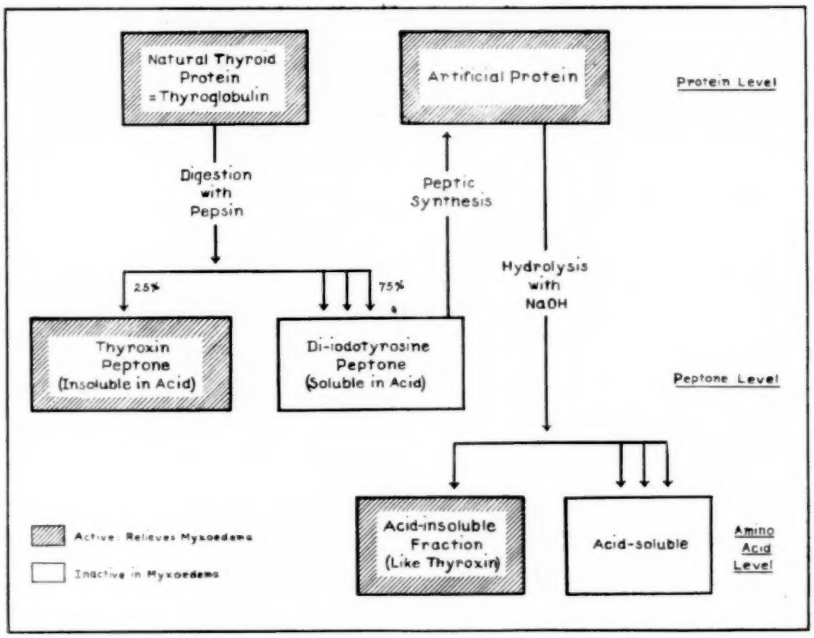

FIG. 1. Diagrammatic representation of the relations between natural and artificial thyroid protein, and their respective cleavage products. Reprinted by courtesy of *Endocrinology* (**20**:801 [Nov.] 1936).

The relation of these substances to each other is shown by diagram in figure 1. The present authors[14] have reported observations on the activity of these two fractions when administered in standard iodine dosage (0.5 mg. iodine) to patients with myxedema. In brief, diiodotyrosine peptone was relatively inert. This is the substance that was used as the starting point in the artificial production of thyroid protein.

It would be proper, also, to use for this purpose an enzymic preparation derived exclusively from thyroid tissue. That normal glands contain proteolytic enzymes (proteases) is evident from postmortem autolysis familiar to those who have worked with thyroid glands. From the spleen, indeed, Hedin[15] has actually prepared extracts that catalyze either protein formation or protein destruction, according to thermodynamic conditions. Experiments on a small scale by one of us (W. T. S.) have indicated that artificial protein can indeed be produced in small amounts through the use of an extract appropriately prepared from thyroid glands. For the relatively large scale production that this work necessitated, one was content to use preparations of commercial pepsin and trypsin. In addition, it has been our particular good fortune to obtain from Prof. J. H. Northrop some of his crystalline pepsin, for which we are deeply grateful.

The method used was initiated many years ago by Danilewski[16] and by Abderhalden[17] in their studies on the natural production of body protein. The technic was further improved by Wasteneys and Borsook,[18] and further modified by one of us (W. T. S.[19]). Briefly stated, the principle is one of reversible reaction in accordance with the mass law:

$$\text{PROTEIN} + H_2O \rightleftarrows \text{PEPTONES}.$$

An enzyme, being merely a catalyst, facilitates the progress of a reversible chemical reaction in either direction. The actual course in any given instance is determined by such conditions as concentration, solubility and temperature. The mathematical treatment of this reaction is covered in a paper by Wasteneys and Borsook.[20] In our particular instance the problem was facilitated by the fact that artificial thyroid protein is relatively insoluble around pH 5.5. Advantage was taken of this fact to remove the end product of the reaction from the system. In this way the reaction was made to proceed further toward completion. The details of this method have been published.[19] Suffice it to say that the product so formed was an albuminous substance, which resembled natural thyroglobulin in many respects. In terms of iodine, the

yields varied from 15 to 40 per cent of the original starting material.

THE COMPOSITION AND PROPERTIES OF ARTIFICIAL THYROID PROTEIN

That the chemical properties of this artificial protein are similar to those of natural thyroglobulin is shown in table 1.

TABLE 1

	Artificial Protein	Natural Protein
Iodine Content	0.2± per cent	0.2± per cent
Nitrogen Content	13 per cent	15 per cent
Isoelectric Point (denatured)	pH 5.5	pH 5.0
Dialysis	80+ per cent not diffusible	95+ per cent not diffusible
Biuret Reaction	++	++
Precipitation by 2 per cent Trichloracetic Acid	++	++
Digestion by Pepsin	++	++
Solubility in 0.01 Normal NaOH	++	++
Apparent Thyroxine Iodine Moiety	13-35 per cent	25± per cent (Boston)
Molecular Weight	18,000±	750,000 (Heidelberger and Svedberg)

A recent review by Alcock[21] points out that no evidence exists that the artificial "proteins" thus far studied are really more complex molecules than the initial peptones. For this reason it is important to emphasize the data that we have obtained on our material. Two types of evidence indicate that our artificial protein is composed of larger molecules than the initial peptone. The first evidence, described in detail elsewhere,[19] is the fact that the initial peptone dialyzes or filters through membranes that hold back the artificial protein. The second type of evidence is based upon measurements of the molecular size by the diffusion method of Northrop and Anson.[22] These measurements indicate that the initial peptone has an average molecular weight of about 6,000 as compared with an average molecular weight of 18,000 for the artificial protein. There is technical ground, moreover, for suspecting that the latter value is low. Until the material can be examined by an ultracentrifuge technic, more accurate values cannot be obtained. It is clear, however, that both types of evidence indicate the conjugation of molecules as the result of enzymic action.

THE BIOLOGIC CHARACTERISTICS OF ARTIFICIAL THYROID PROTEIN

Of paramount interest is the biologic effectiveness of this artificial product as compared with the natural thyroid hormone. This point may be tested in clinical myxedema by comparative assays of the two proteins and their respective hydrolytic products. Actual curves showing the rise in the basal metabolic rate of individual patients with myxedema when fed this artificial protein are shown in figure 2. They show an average daily

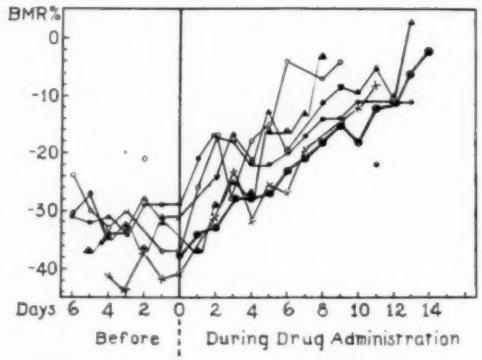

FIG. 2. The calorigenic responses of five patients with spontaneous myxedema to daily oral doses of artificial protein (containing 0.5 mg. of total iodine). Two patients received protein made from diiodotyrosine peptone that had undergone ultrafiltration (solid lines and solid triangles); the other three received protein made from diiodotyrosine peptone that had been freed of thyroxine peptone and thyroglobulin by the usual chemical procedures. The heavy solid line, also present in figure 3, is the standard response to daily doses of 1.0 mg. of natural thyroxine polypeptide (0.5 mg. total iodine). Reprinted by courtesy of *Endocrinology* (**20**:801 [Nov.] 1936).

rise in metabolism of 2.6 per cent. The homologous effect produced by natural thyroid colloid is shown in figure 3; the average daily response

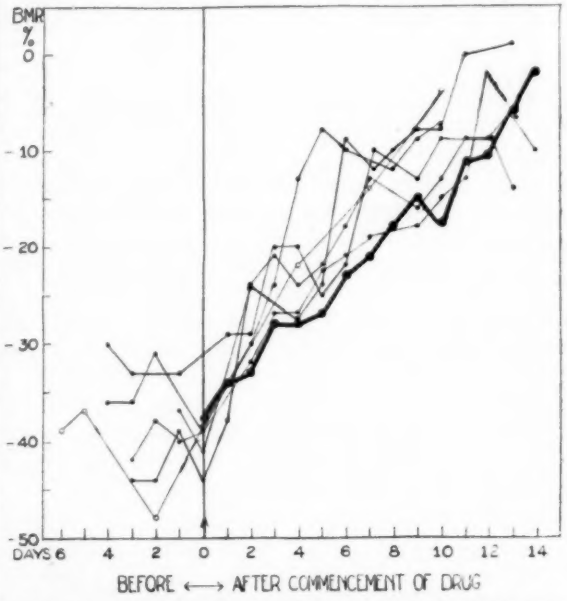

FIG. 3. The calorigenic responses of five patients with spontaneous myxedema (light solid lines and solid circles) and one patient with cretinism (light solid lines and open circles) to daily oral doses of thyroglobulin (containing 0.5 mg. of total iodine). Three patients received thyroglobulin derived from human, colloid goiters, and the other three received thyroglobulin derived from human hyperplastic glands ("toxic").

of 2.8 per cent is essentially identical.

In addition, as shown in figure 1, the artificial protein could be hydrolyzed into two fractions analogous to the two peptone constitu-

ents of natural thyroglobulin previously referred to.[13] These likewise showed analogous hormone activity.

An interesting feature of the biologic activity of natural thyroid protein is a discrepancy between assays made in myxedematous human beings and those made in normal animals. The former appear to react to the total iodine, whereas the latter seem to respond only to the thyroxine fraction. It was suggested that the reproduction of this discrepancy would be impressive evidence as to the similarity of the artificial and natural proteins. Through the courtesy of Drs. Palmer and Leland,[28] we were able to put this question to test. The results of their work will be reported in detail elsewhere, but we quote from their findings as follows: composite curves of metabolism in normal guinea pigs were obtained, "one representing the total iodine equivalent, and the other the approximate thyroxine iodine equivalent. . . . The activity obtained correlates well with the thyroxine present." In short, the normal animal seems to respond only to the thyroxine-like fraction of the artificial protein just as it does in the case of natural thyroglobulin, and the discrepancy between thyroidless man and the normal animal is reproduced.

DISCUSSION

An artificial protein, which possesses marked metabolic activity in human myxedema, has been synthesized from a relatively inert material by enzymic action. Furthermore, this artificial protein has many of the quantitative chemical and biologic characteristics of the natural protein.

The possible presence of traces of thyroxine impurity in diiodotyrosine preparations made it essential to control carefully this source of error. This was done by the following sort of arithmetical reasoning. In one instance, an amount of initial peptone was taken such that, by guinea-pig assay, fifty standard doses of iodine might conceivably have contained one dose of thyroxine impurity. The artificial protein obtained therefrom, however, was actually sufficient for seven doses, in treating human myxedema. On the other hand, seven doses of the original material produced no effect in human myxedema. A detailed account of this quantitative control is published elsewhere.[24]

These studies throw light upon the physiologic changes induced in the thyroid when iodine is administered to a patient whose thyroid is poor in colloid. The immediate effect is the storage, in large amounts, of newly-formed thyroid protein in the thyroid follicles. The present observations suggest that a simple enzymic synthesis may be the mechanism by which this new protein is formed, with the proteases of the body or thyroid gland acting as the catalysts. They suggest, in short, that the therapeutic effect of iodine is indirectly a mass-law phenomenon. This deduction is based on the hypothesis that when inorganic iodine is administered, it first appears in a chemical form similar to diiodotyrosine peptone. Were this true, diiodotyrosine peptone might be considered as a prohormone, that is, a precursor, in the same sense that carotin is termed a provitamin with reference to vitamin A, or ergosterol with reference to vitamin D. Interpreted in this light, the effect of iodine therapy is, first, to favor the synthesis of diiodotyrosine peptone, and when this accumulates, to lead to the synthesis and deposition of hormone rather than to its release.

CONCLUSIONS

Biologically active thyroid protein may be obtained from relatively inactive diiodotyrosine peptone by enzymic action according to the mass law. Since the initial effect of iodine therapy in hyperthyroidism is the accumulation of diiodotyrosine in the thyroid gland, it is reasonable to assume that the depleted stores of hormone in the gland are rebuilt from this precursor by thyroid enzymes. The relief produced by iodine is visualized as a mass-law phenomenon, which dams back the flow of hormone outward from the gland, and causes it to accumulate in the thyroid follicles.

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PRINCIPLES OF PARATHYROID SURGERY*

BY EDWARD D. CHURCHILL, M.D.†

THE treatment of hyperparathyroidism by surgery is a direct and straightforward procedure entirely in accord with basic principles of endocrinology. It is based fundamentally upon the assumption that the ablation of hyperfunctioning tissue reduces the hormonal secretion of the gland, thereby correcting the disturbed metabolism of the disease. I shall outline briefly the problems that have been encountered in attempting to perfect a precise and effective treatment for hyperparathyroidism.

Diagnosis. Before any therapeutic measure can be developed or scientifically assayed, a positive diagnosis in the individual case is imperative. Objective measurements of the pathologic state and the change effected by treatment are essential. Violation of these simple precepts has created confusion in every field of medicine, and particularly at the moment, clouds the clinical applications of the science of endocrinology. In well-established cases of hyperparathyroidism the diagnosis is certain, and objective measurements of therapy are readily available. This fact, in itself, accounts for the sound progress that has been made in the recognition and treatment of the disorder during the past decade. The diagnosis of the disease has, on the whole, been more precise and certain than the scalpel of the surgeon attempting to cure it.

Surgical Technic. The technical aspects of the operation are fundamentally simple. The neck is a readily accessible region of the body and can be safely approached by any surgeon trained to employ a deliberate and painstaking technic. Technical barriers such as those which delayed the development of surgery of the brain or surgery of the thorax are nonexistent.

Anatomy. The anatomic difficulty confronting the surgeon centers on the fact that the parathyroid apparatus is not a single organ, but is divided into a number of discrete units. Careful anatomic dissections may yield from two to six or more parathyroid bodies in a single individual. The typical number is four.

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The location of the parathyroid bodies is subject to wide variation. While commonly pictured as lying on the posterior surface of the thyroid gland, they are so frequently found at a distance from the gland that such a position can scarcely be termed "aberrant." The space bounded *posteriorly* by the fascia covering the longus colli muscles, *anteriorly* by the middle cervical fascia, *superiorly* by the pharynx and extending *inferiorly* for an unknown distance into the mediastinum may be considered as the normal region for the distribution of parathyroid tissue.

Given this region for surgical dissection, the problem resolves itself into recognizing normal or abnormal parathyroid bodies in the gross. Painstaking dissections in the autopsy room, checked by immediate histologic examination, have made the identification of parathyroid tissue certain, once it is disclosed.

Pathology. The descriptive morbid anatomy of the parathyroids was exceedingly fragmentary at the time of the beginning of medical and surgical interest in their abnormal function. They had rarely been looked for or examined in routine postmortem examinations. At the outset the gross size of the gland was taken as an index of a pathologic state. Through the study of specimens removed on this basis the cellular pathology of the parathyroids has been systematized by Castleman and Mallory. Gross size is, at the present time, not acceptable in itself as a criterion of normal or pathologic function, but must be interpreted in conjunction with the histologic findings. It is quite possible that certain hyperfunctioning glands have been overlooked at operation or autopsy because their gross dimensions lay within the limits of normal variation.

Microscopic studies of the tissue have established two types of pathology that are of great practical importance in surgical therapeutics. Six out of thirty-five cases of proved hyperparathyroidism have shown a hyperplasia of all parathyroid bodies. In the remaining cases the pathology has been localized to one or, rarely, two glands. The type of pathology cannot be predicted in an individual case by

the clinical findings, but is disclosed at operation.

At the present time these pathologic entities are designated hyperplasia and neoplasia, respectively. By neoplasia is meant a focal proliferative process, commonly termed an adenoma. The cellular change may be so sharply localized that it is limited to only a portion of a single parathyroid body, normal gland parenchyma forming a marginal fringe in cross-section. Hyperplasia stands in sharp contrast to the adenomas, as all glands are uniformly involved in a proliferative change that produces a constant histologic picture.

The hypothesis that assigns a dual nature to these proliferative changes is one of basic biologic interest and finds a parallel in other glands of internal secretion. The parathyroids provide a peculiarly clear and unique exposition of the problem because of the anatomic division of the glandular tissue into four discrete and often widely separate units. Diffuse and uniform hyperplasia of all units suggests an external or humoral stimulus; on the other hand, a focal and discrete collection of cells, limited, at times, to a portion of one unit, speaks for a local and autonomous factor underlying the pathologic process.

In surgical therapeutics the adenoma offers an opportunity to extirpate completely the offending tissue. In diffuse hyperplasia the parathyroid bodies are considered as constituting a single hyperfunctioning gland, and a subtotal resection of the entire apparatus is necessary to reduce its function. By the trial and error method it has been found that a modicum of parathyroid tissue roughly equivalent in size to a single normal parathyroid body is sufficient to meet the needs of a normal individuai.

Physiology. Operations planned entirely upon regional anatomy and pathology often lead to disaster, unless tempered by consideration of more subtle and remote physiologic principles. The tetany that follows a complete removal of all parathyroid tissue is familiar in surgery as a disaster incident to thyroidectomy. In parathyroid surgery this hazard exists, but should occur only as an accident that can be guarded against by painstaking technical methods.

Life-endangering tetany may be encountered, however, even though sufficient tissue is preserved to meet the needs of a normal individual. The sudden reduction of parathyroid hormone may initiate such a rapid movement of calcium salts into the skeleton that the blood level is lowered to a dangerous degree. The rate of deposition in the bones appears to be governed by the amount of depletion of the skeletal reservoir and, particularly, the state of activity of the osteoblastic tissue. Fortunately, an index to osteoblastic activity is found in the concentration of the enzyme, phosphatase, in the blood. Patients showing a high concentration of phosphatase should be subjected to graded operations removing only a part of an adenoma at one time, in order to avoid the hazards of this tetany of the recalcification period.

Results of Treatment. In judging the results of surgical treatment a differentiation must be made between reversible and nonreversible pathologic changes that have occurred during the course of the disease. The decalcification of the skeleton is rapidly corrected. The giant cell tumors regress, and ununited fractures are repaired by calcification of the callus. Cystic areas may remain indefinitely, although they are strengthened by the deposit of calcium. In long-standing cases with skeletal deformity, permanent crippling will remain.

Certain insidious symptoms of the disease, such as general muscular weakness, which may be linked with the abnormal level of blood calcium, often disappear with dramatic rapidity.

The renal complications induced by hyperparathyroidism are of grave importance to the individual patient. Certain of the pathologic changes that result from a long-continued, high concentration of calcium in the urine appear reversible. Discrete calculi may be removed surgically at a subsequent date. The renal manifestations of the disease, however, constitute the real menace to life in the average case, and demand an early recognition of the disease and the immediate institution of proper treatment.

FACTORS GOVERNING THE CALCIUM EQUILIBRIA IN THE BODY*

BY A. BAIRD HASTINGS, PH.D.†

THE chemist and physician, Van Helmont, stated three hundred years ago that he would devote his life to the study of dropsy, epilepsy, calculi and gout. He seems to have chosen his problems well because we still have them with us. Indeed, save for the symposium being held today on infectious diseases, he would feel at home at any one of our conferences. When a chemist of today studies "calcium equilibria in the body," he hopes that some of the information which he obtains will aid in enlarging our understanding of why calculi form; and there are those who think that such information may

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even be of service in the study of dropsy and epilepsy.

Be that as it may, in any discussion concerning the growth of bones, teeth and pathologic calculi on the one hand, or the absorption of calcium salts from such deposits on the other, the part played by the underlying physicochemical laws that govern the precipitation and solution of salts is not to be ignored. The part that certain of the endocrine glands play, directly or indirectly, in modifying the physicochemical factors is recognized as highly important; but the detailed chemical description of this role is beyond our present knowledge. It is my present purpose to state the nature of these underlying physicochemical laws and to outline the role which they may conceivably play and the extent to which they are useful in studying calcium and phosphate metabolism in the body.

The present discussion will deal with the Ca^{++}, $PO_4{\equiv}$ and $CO_3{=}$ concentrations of body fluids, the calcium, inorganic phosphate and carbon dioxide content of bone, and the relation between the composition of the solid phase and the ionic concentrations of the liquid phases.

When one undertakes the study of the solubility of the calcium salts of bone, one has great difficulty in determining the nature and composition of this solid material. True, one can analyze bone for its Ca, PO_4 and CO_2 content, but this is not enough, as experience has sadly shown. An example of such an analysis, made by Dr. Logan, is shown in the accompanying table. From such data, one might reasonably conclude that the calcium salts of bone were a mixture of 12 per cent $CaCO_3$ and 88 per cent $Ca_3(PO_4)_2$. Expressed on a molar basis, such a result could also be interpreted as indicating that bone is a mixture of approximately 2 mols of $Ca_3(PO_4)_2$ and 1 mol of $CaCO_3$.

TABLE 1

Analysis of Bone			By Calculation		
Ca	PO_4	CO_2	$CaCO_3$	$Ca_3(PO_4)_2$	$\frac{Ca_3(PO_4)_2}{CaCO_3}$
mM.	mM.	mM.	%	%	
gm.	gm.	gm.			
5.95	3.47	0.80	12	88	2.1

The State of the Calcium Salts of Bone.—Bone is not a mechanical mixture of $Ca_3(PO_4)_2$ and $CaCO_3$ such as one might prepare by grinding the two salts together in a mortar. Indeed, there is grave doubt that the pure substance, $Ca_3(PO_4)_2$, can exist in equilibrium with an aqueous solution. This became clear when Taylor and Sheard, and subsequently others, investigated the structure of bone salts by means of x-ray spectrograms. These demonstrated that bone is crystallographically identical with the mineral, dahllite, a carbonate containing apatite. In such a compound, calcium carbonate, as such, apparently does not exist. It has lost its identity. No evidence was found that indicated the presence of either crystal form of $CaCO_3$, calcite or aragonite, nor was there any evidence for the presence of calcium diphosphate, $CaHPO_4$. Indeed, the x-ray spectrogram of bone seems not to be significantly different from that of any apatite mineral which has the general chemical formula $CaX \cdot nCa_3(PO_4)_2$, where X may be F, OH, or CO_3, and n usually varies between 2 and 3.

This illustrates, then, the first difficulty encountered in studying the solubility of calcium salts of bone in biologic fluids, namely, that there has been in the past great uncertainty as to what calcium salts one should actually use in studying the solubility of bone salts in the laboratory.

But to determine the proportion of the constituents of bone is not enough. It is important to know whether one is dealing with a definite chemical compound whose formula may be written 1 mol of $CaCO_3$ 2 mols of $Ca_3(PO_4)_2$, or whether the $CaCO_3$ is present in some other form. Experiments recently carried out in the biochemical laboratories of the Harvard Medical School by M. A. Logan and H. L. Taylor have already shed some light on this question. They prepared solutions that were supersaturated with respect to $Ca_3(PO_4)_2$ but undersaturated with respect to $CaCO_3$, and analyzed both the solution and the precipitate, which formed at various intervals up to 28 days. They found that the precipitate, formed within the first 15 minutes after the solutions had been mixed, contained a considerable amount of CO_2, and that the proportion of CO_2 in the precipitate gradually increased with continued equilibration until, after 28 days, it had doubled in value. This is the more remarkable because, as I said, the solubility product of $CaCO_3$ was not exceeded during the course of the experiment, and one would *not* have anticipated the inclusion of $CaCO_3$ in the precipitate.

These results can be interpreted as showing that the calcium salts of bone consist of a solid solution of $CaCO_3$ in $Ca_3(PO_4)_2$ and that the constant value, 2, of the ratio of $Ca_3(PO_4)_2 : CaCO_3$ found in adult bone is to be attributed to the constancy of the surrounding fluid environment and to the efficiency with which the fluid is equilibrated with the calcium salts in the organism.

The Ca^{++}, $PO_4{\equiv}$ and $CO_3{=}$ Concentrations of Body Fluids.—I shall now review briefly what we know of calcium ion concentrations in biologic fluids. Since a certain product of the ions, Ca^{++} and $PO_4{\equiv}$, must be exceeded for calcification, regardless of the physiologic factors

that influence them, it becomes of importance to have knowledge of the concentration of these ions in biologic fluids. Actually, the knowledge that we desire is the concentration of Ca^{++} and $PO_4{\equiv}$ ions at the *site* of calcification. Since this is beyond our technical capacities at the present time, investigators have had to content themselves with the study of such fluids as blood serum, lymph, cerebrospinal fluid, edema fluid, and the like.

Much work has been done, and a variety of technics employed in the study of this phase of the problem. Considerable interest in the calcium ion concentration of body fluids has arisen, not only because of its importance in calcification, but also because of its importance in problems involving neuromuscular irritability and the manner in which certain endocrine glands—notably the parathyroids—influence these ionic concentrations.

By careful standardization and a thorough study of the behavior of the heart of the frog in different ionic environments, McLean and the author were able to utilize this biologic indicator for the estimation of calcium ion concentrations in biologic fluids, where those of us previously utilizing strictly chemical procedures had failed. Although there is still no chemical method for calcium ion determination that can be applied to biologic fluids, the validity of the use of the frog heart as a calcium ion indicator has been confirmed in simple solutions containing proteins and citrate where chemical methods are applicable.

Using this method, it has been found that the calcium ion concentration of nonprotein containing fluids, such as transudates and cerebrospinal fluid, is practically identical with the total calcium concentration. That is, there is no significant amount of an nonionized calcium compound to be found in such fluids. Under normal conditions, the calcium ion concentration and the total calcium concentration may be regarded as identical and equal to approximately 4.5 mg. of Ca per 100 cc. or 1.1 millimols per liter.

When one studies blood serum, however, one finds approximately 50 per cent of the total calcium present in nonionized form. This is due to the formation of a calcium protein salt, which is only partially ionized. The relation between the ionic and nonionic calcium can be expressed by a simple mass-law equation:

$$\frac{Ca^{++} \times Prot^{=}}{CaProt} = 10^{-2.22},$$

where $10^{-2.22}$ is the ionization constant of the calcium protein salt.

The validity of this equation has been demonstrated over a wide range of protein and calcium concentrations and in a variety of pathologic conditions—notably nephritis, rickets, hyperparathyroidism and hypoparathyroidism. No evidence has been found that would indicate the presence of more than 5 per cent of the serum calcium in a diffusible, nonionized form. It is, therefore, possible, knowing the total protein and total calcium concentrations, to calculate the calcium ion concentration of a particular serum.

I shall not describe the calculation of the $CO_3^{=}$ and $PO_4{\equiv}$ ion concentrations other than to state that the $CO_3^{=}$ ion concentration of serum can be calculated from the mass-law equation of carbonic acid, when the pH and total CO_2 are known; and $PO_4{\equiv}$ concentrations can be calculated from a similar though more complex equation when the pH and total inorganic phosphate are known.

We have at our disposal means of calculating the ion products of biologic fluids, $(Ca^{++})^3 \times (PO_4{\equiv})^2$ and $(Ca^{++}) \times (CO_3^{=})$. For normal serum, $(Ca^{++})^3 \times (PO_4{\equiv})^2 = 10^{-23.5}$ and $(Ca^{++}) \times (CO_3^{=}) = 10^{-6.7}$.

Relation between Ion Products of Fluids and Bone Salts. The question immediately arises as to whether these biologic fluids are supersaturated, in equilibrium or undersaturated, with respect to the calcium salts of bone. This could be answered if one knew how large the ion product must be to cause precipitation of these salts, and how small it must be for their solution.

Logan and Taylor have shown that when aqueous solutions have an ion product, $(Ca^{++})^3 \times (PO_4{\equiv})^2$, much greater than $10^{-23.5}$, $Ca_3(PO_4)^2$, containing $CaCO_3$, will precipitate. Furthermore, when the amount of precipitate exceeds 150 mg. per liter, precipitation will continue until an ion product of 10^{-27} is reached. Dr. Audra Browman found similar ion products for serum and cerebrospinal fluid when they were shaken with relatively large quantities of bone. When the ion product is *less* than $10^{-23.5}$, no precipitate will spontaneously form, even if it is seeded with a minute amount of $Ca_3(PO_4)_2$ or bone salts. The failure of solid $Ca_3(PO_4)_2$ to form from biologic fluids unless the Ca^{++}, $PO_4{\equiv}$ ion product is increased above its usual normal value of $10^{-23.5}$ is thus understandable.

Blood serum, however, is not in equilibrium with the calcium salts of bone as they finally exist in mature bone. As has been stated, when $Ca_3(PO_4)_2$ has taken up the maximum $CaCO_3$ of which it is capable, under the conditions presented to it by biologic fluids, that is, when

$$\frac{Ca_3(PO_4)_2}{CaCO_3} = 2,$$

the solubility product of the $Ca_3(PO_4)_2$ is 10^{-27}. This means that the ion product of biologic fluids would have to be less than 10^{-27} to bring about the solution of the calcium salts of bone.

Furthermore, if body fluids are shaken with bone, there will be precipitation of calcium salts until the ion product, $(Ca^{++})^3 \times (PO_4\equiv)^2$, reaches 10^{-27}. This has been found to be the case when one uses a relatively large amount of solid, and leads one to the conclusion that body fluids, such as blood plasma and transudates, are supersaturated with respect to the calcium salts of bone as they exist in their final form. This is the conclusion previously reached by Holt, LaMer and Chown.

On the other hand, one cannot conclude from such data that body fluids are supersaturated with respect to the form of calcium phosphate which *first* precipitates from such solutions. It is well established that body fluids in vivo have, under normal conditions, a remarkably constant calcium ion concentration. When the calcium content of the body fluids is reduced, as, for example, by repeated perfusion with blood from which the calcium has been removed, the calcium content of the circulating plasma is quickly restored to its normal value. On the other hand, when the calcium is raised above normal, the calcium concentration of the plasma rapidly decreases to its original value. Whether or not there is a true equilibrium in the thermodynamic sense, there is, at least, a steady state that results in the maintenance of an effective physiologic equilibrium. Further experiments by Logan and Taylor are of interest in this connection. Salt solutions equilibrated for 8 days with varying amounts of bone powder not exceeding 100 mg. per liter were found to have ion products more than 10^{-27}. It is of interest that with the smallest amount of solid, 10 mg. per liter, an ion product was found not far from that of normal biologic fluids.

Taking, then, into consideration the chemical facts that have been established regarding the system, BONE SALTS : BODY FLUIDS, the following conclusions may be drawn:

1. The inorganic phase of mature bone is essentially $Ca_3(PO_4)_2$ containing calcium carbonate in solid solution, approximately in the proportion of 1 mol of $CaCO_3$ for 2 mols of $Ca_3(PO_4)_2$.
2. The fluid in immediate contact with this inorganic phase presumably has an ion product, $(Ca^{++})^3 \times (PO_4\equiv)^2$, of 10^{-27}. If it becomes less than 10^{-27}, $Ca_3(PO_4)_2$ will go into solution. If it becomes greater than 10^{-27}, more Ca, PO_4 and CO_2 will be removed from solution.
3. For the initiation of precipitation from a fluid, the ion product $(Ca^{++})^3 \times (PO_4\equiv)^2$ must exceed the value $10^{-23.5}$. Once started, the formation of solid will continue until an ion product of 10^{-27} is reached, provided the proportion of solid to fluid is greater than 150 mg. per liter. The precipitate formed from body fluids will initially consist of $Ca_3(PO_4)_2$ containing little $CaCO_3$; with time, however, the composition of the precipitate will change until, in mature bone, it consists of tertiary calcium phosphate and calcium carbonate in the approximate ratio of 2 to 1.
4. On the other hand, no precipitate will spontaneously form in fluids, such as blood plasma, not in contact with bone salts, provided their ion product does not exceed $10^{-23.5}$.

In the foregoing discussion, I have limited my remarks to those physicochemical considerations that are believed to be pertinent to the problem of calcification. The biologic factors, such as the absorption and excretion of calcium and phosphate, the activity of the enzyme, phosphatase, the role played by vitamins and hormones, and local metabolic processes that affect the pH of the fluids, are of great importance. Unfortunately these are subjects that cannot be discussed from a chemical standpoint at this time.

Much remains to be done; but we hope that the stage is now set. We know, with greater certainty than before, the chemical composition and the essential properties of the components of the system with which we are working. Why body fluids are apparently supersaturated by one criterion, and in equilibrium with bone by another, is no longer such a mystery. Shall we now be able to push our frontier of quantitative knowledge back to the actual site of calcium salt deposition? That is a question that further experiment, alone, can answer.

THE FREQUENCY, RECOGNITION AND TREATMENT OF CHRONIC SUBDURAL HEMATOMAS*

BY GILBERT HORRAX, M.D.,† AND JAMES L. POPPEN, M.D.†

IN a recent paper Munro[1] has rightly called attention to the fact that all posttraumatic subdural hematomas, whether acute or chronic, represent merely different stages in the same underlying process, and little, if anything, can be added to his excellent portrayal of the recognition and handling of these cases. The great majority of Munro's patients had subdural clots that ordinarily would be classified as "acute" or "subacute" following known head injuries for which they were being treated. From the purely clinical aspect, however, it is our belief that the group of cases that have long been termed "chronic subdural hematomas" — the old pachymeningitis hemorrhagica interna—represents a condition that calls for certain comment, particularly in regard to its diagnosis and treatment.

In the first place, such trauma as has occurred in patients with the type of clot in question has been either slight or long since forgotten. As a rule, the physician who sees the patient originally — if, indeed, the injury is severe enough to consult a doctor—either dismisses him as well or at least sufficiently improved so that no further treatment at the time is indicated. Subsequently the patient consults a neurologist, psychiatrist or neurosurgeon because of difficulties that appear to be in the sphere of one or the other of these specialties. It is, therefore, very largely a question of the differentiation of intracranial tumor, cerebral arteriosclerosis, syphilis, or a possible subdural hematoma for which these patients eventually seek help, although in some instances the question of posttraumatic headaches may be in the foreground.

The type of lesion with which we are concerned is the long-recognized, large, completely encapsulated, usually liquefied, blood clot that occupies the space between dura and brain surface and almost invariably covers two thirds to three quarters of one cerebral hemisphere. It is not uncommon to find bilateral clots of this character, and why they do not give more profound neurologic signs, or at least why such signs or symptoms appear only after a fairly long period of time is most puzzling. The blood sac often averages an inch in thickness throughout its greater portion, and, of course, compresses the hemisphere to a marked degree.

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The three points which we wish to emphasize in this communication will be taken up in order.

1. *Frequency.* The condition is now recognized much more frequently than was the case even 5 or 10 years ago. This feature was referred to by the authors in an article in the *Surgical Clinics of North America* in 1935.[2] To summarize, it may be said that in 1925 Putnam and Cushing[3] could find only 5 cases of subdural hematoma in the records of the Peter Bent Brigham Hospital for the previous 9 years, and, even in 1935, Frazier[4] reported but 6 cases for the years 1932 and 1933 from his clinic at the University Hospital, Philadelphia. On the other hand, a rather large number of articles by various neurosurgeons who had treated a relatively small series of these cases appeared between 1929 and 1935 and showed that those concerned with the surgical treatment of this disorder were recognizing it with increasing frequency. At the time of our previous paper, to which we have referred, 12 patients having chronic subdural hematomas had come under our care during the previous year and a half. The series has now risen to 18 in a total period of 2 years.

That this figure represents a striking increase in the number and frequency of chronic hematomas in one clinic may be seen from the references just cited, and also by a comparison of this paper with a recent article by Furlow[5] in which he reports 16 cases over a period of 15 years from the clinic of Dr. Ernest Sachs in St. Louis. Our relatively large series of patients with clots of the chronic, encapsulated variety is due, we believe, to two principal factors: first, because as "brain tumor suspects", they have been sent to us for diagnosis and, secondly, because in an increasing number of cases previously diagnosed as "cerebral arteriosclerosis", "old encephalitis", "posttraumatic headache" and the like, we have made sure that such diagnoses were correct by doing simple bilateral taps over either side of the skull and, thus, quite unexpectedly in many instances, have disclosed and evacuated the lesion in question.

2. *Recognition.* As set forth in the previous paragraph, the surest, and, in our experience, one of the most frequent ways in which chronic subdural hematomas have been recognized is by visualization after bilateral, small burr openings have been made in the skull. It may well be asked, "What are the indications for these tappings?" or "When is one justified in subjecting a patient even to such a minor surgical procedure?" There is no single or simple answer to these questions, as much must depend

upon the judgment and experience of the surgeon who is consulted. Several distinct classes of patients may be cited, however, for whom it is essential to take this final step in making a correct diagnosis.

In the first group belong those patients whose histories suggest strongly the presence of a hematoma. In such cases there is a definite story of head trauma, with or without a "latent period" of freedom from symptoms, after which headaches begin, continue and are likely to become extremely severe. As has been suggested, the "latent period" may not be present at all, but during the first week or two after injury the patient is perhaps so uncomfortable in many ways that it is only after an appreciable interval that he realizes that persistent headache is or has been present. On the other hand, an interval of weeks or even months may elapse before the patient begins to complain of headaches. Other symptoms after a known head injury likewise suggest a hematoma. Chief among these are vague, sometimes transitory, but usually increasingly marked, mental aberrations. Sometimes these are trifling and are noticed only by near relatives but, as time goes on, they become more and more apparent. Indeed, it is not at all infrequent that patients in this category are first taken to a psychiatric clinic, whence, if their condition is suspected and then verified by tappings, they emerge with normal mentality.

A second class of patients is represented by those that are thought to have a brain tumor. There may be a history of head trauma but this is also frequent in patients that actually have tumors. The clinical findings in this group may include apparently localized phenomena, such as weakness and sensory changes over one side of the body, aphasia and unilateral increase in the deep reflexes. Choked disks are not common, but they may reach a degree of 2 to 3 diopters.

One other group may be mentioned, namely, those patients that may have had occasional headaches and perhaps indefinite mental symptoms, curious and unaccountable lapses into deep drowsiness or even coma, but no objective evidences otherwise of increased intracranial pressure. These unfortunates are likely to be diagnosed as cases of "cerebral arteriosclerosis", "cerebral atrophy" or possibly "postencephalitic state". The fundi are normal, and in many instances, no definite history of injury to the head can be obtained.

In all of the above groups and in those that simulate them closely, it is essential to be sure whether one is dealing with an encapsulated subdural clot, for there could be no greater calamity than to have a patient die or be put into a hospital for mental disorders when his condition is curable. The diagnosis can be arrived at with certainty and with almost no risk by making bilateral small openings in the skull and by inspecting and incising the dura in the manner that will be given in detail later. Some form of intracranial lesion is known to exist in the second group of patients that have been listed and they would naturally have a complete investigation, but in the other two groups, if tappings are not made, the burden of a questionable diagnosis is distinctly upon the physician who assigns such a patient to the discard of "cerebral arteriosclerosis", "posttraumatic state" or some form of mental disorder requiring institutional care.

One word of explanation should be made here. It is not to be inferred that all patients with some degree of headache following a head injury are candidates for cerebral taps. Judgment must be exercised to the highest degree in these cases, but, if there is any real question of a subdural hematoma, an encephalogram may be performed instead of the taps. If no hematoma exists, the air insufflation may benefit the headaches, but if a clot is present, it will be revealed by the x-ray and may be evacuated by a subsequent tap.

3. *Treatment.* If it is decided that a patient with symptoms corresponding to one of the foregoing groups is a candidate for exploration, the procedure is carried out in the following simple manner.

An area 10 cm. square is shaved over the parieto-occipital region of the scalp, and, if the patient is not drowsy or in coma and if he shows no evidence of increased intracranial pressure, a small dose of morphin and scopolamin may be administered. The patient is then placed on his back on the operating table with the head on two sandbags in such a manner that the neck is flexed forward, a position that gives good access to the shaved area. After the usual preparation of the scalp with bichlorid of mercury and alcohol, a small infiltration with novocain is made about 4 cm. lateral to the median line on either side, and scratches are made in the sagittal plane over both infiltrated areas to indicate the lines of incision (figure 1). The patient is then draped, the incisions are made along the scratch lines down to the bone, the periosteum is separated from the bone and the wounds are held open with mastoid retractors. Burr openings are then made in the bone on either side with whatever type of drill the operator may prefer (figure 2).

When the dura is thus exposed over both hemispheres it should be carefully inspected. If it is of the normal white or pinkish white appearance, no clot, as a rule, is present. To be perfectly sure, however, the dura should be incised and a normal cortex demonstrated. At this point, the question arises as to whether a clot may exist apart from the area that is explored. The answer is obtained by introducing

needles into the ventricles, which normally are found directly inward at a depth of about 4½ to 5 cm. from the dura, by emptying them and by filling them with air and subsequently taking

FIG. 1. Photograph to show area of shaved scalp in the occipitoparietal region, together with the scratches that indicate the lines of incision.

x-rays. These will show compression or distortion of one or both ventricles according to whether unilateral or bilateral hematomas are present. It is most unusual, however, to have

FIG. 2. Photograph of the draped operative field, the incisions held apart with retractors, and the burr holes in the skull. The incisions are in the occipitoparietal region, on either side of the median line.

an encapsulated clot that covers such a small area that it is missed by a burr opening in the position given. The majority of hematomas occupy an area equal to or greater than that shown in the diagrammatic sketch (figure 3). In no instance in this series was a hematoma identified by ventriculography when the usual taps had been negative.

If the lesion is present on one or both sides, the dura is much darker than normal and of a

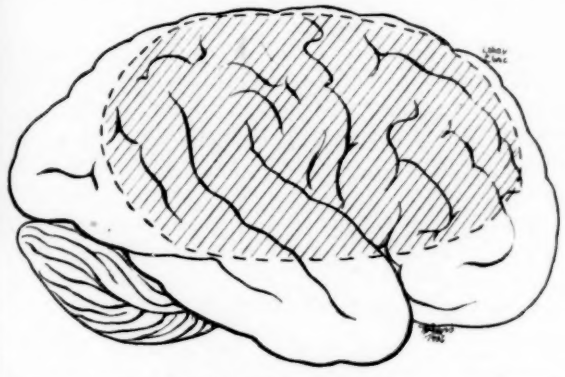

FIG. 3. Diagrammatic sketch to show the usual approximate area of a subdural hematoma.

greenish or bluish black color (figure 4). Upon incising the dura the greenish brown membrane that forms the outer capsule of the hematoma is revealed. One should make sure that a large vein is not presenting, as sometimes this simulates a hematoma capsule. The membrane is then incised, and, if the clot is partially or wholly liquefied, its contents, brownish black in color, pour out, or better, are sucked out with a catheter attached to a syringe, so that the amount may be measured. After all available fluid has been obtained in this way, the catheter should be gently introduced far within the great cavity, and the latter should be washed repeatedly, but very carefully, with normal saline solution. In this way a certain amount of soft, partially organized material, as a rule, will be detached and come out with the washings.

In our experience it has not been found necessary to make more than one opening over each hemisphere, as the desired result can be assured by the method just described, except in the case of largely or completely organized hematomas. In these cases it is essential to turn down a bone flap over the side that is involved and to extract the lesion in whatever way seems suitable. It should be borne in mind, however, that it is necessary to extract only the major portion of the clot and its capsule. When the latter is adherent to the delicate veins that connect the cortex with the longitudinal sinus, it should be allowed to remain, as it will be absorbed, and attempts to remove it might tear off a vein and result in a serious hemorrhage.

In the usual procedure by the simple burr openings, the wounds are closed with fine silk in two layers without drainage of any kind. On several, sometimes many, succeeding days

we have been accustomed to introduce a ventricular needle through the closed wound and, when the cavity is reached, further material from the clot, which was not obtained at the original operation, is aspirated. The amount becomes less each day until little or none is present. By this time the hemisphere has expanded to fill up the cavity from which material of the clot was evacuated.

It is to be realized, of course, that in advocating the exploratory punctures just described a certain number of negative procedures will result. We feel, however, that with careful judgment the latter may be held down to a minimum, and certainly with the greatly increasing number of hematomas that have been disclosed, this simple measure seems more than justified. In proper hands, the taps may be carried out quickly and safely and, in many instances, they represent the only possible means of making a diagnosis as well as being the usual method of effecting a cure.

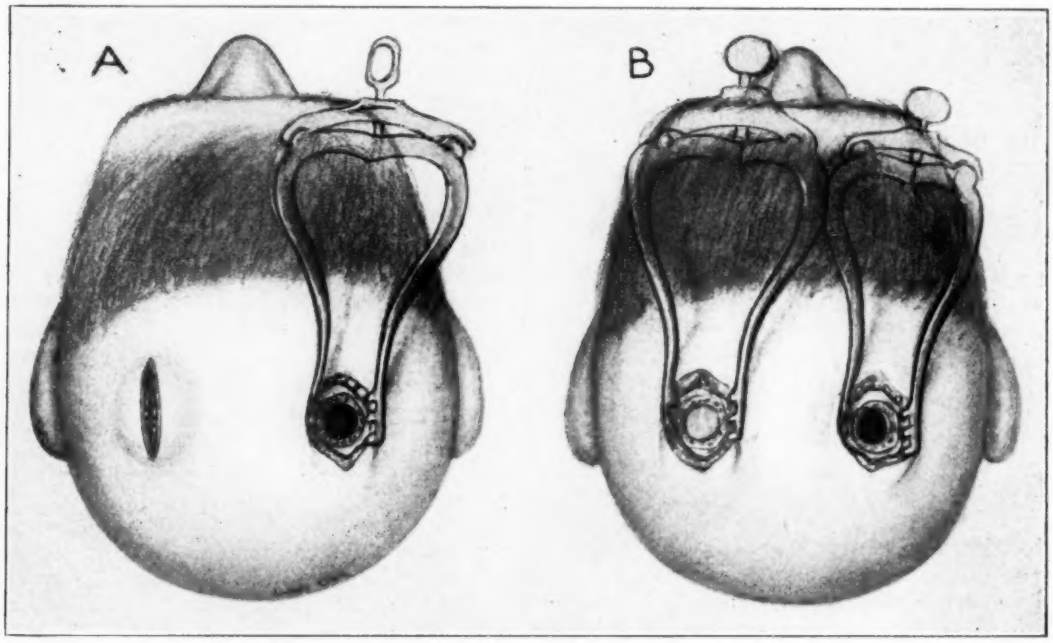

FIG. 4. To show (A) the site of skin incision and burr opening with an underlying hematoma. (B) Normal appearance of the dura (left) compared with the discoloration from a hematoma (right).

RESULTS

There were no postoperative deaths among the 18 patients in this series. By this it is meant that no patient died during the time he was in the hospital, no matter what the length of that period may have been. Most of the patients were discharged in from 10 days to 3 weeks, but in one or two instances the hospital stay was considerably longer. Only 1 patient has died, subsequent to discharge from the hospital, and, so far as could be told from the necropsy, his death was not due to development of another hematoma or to any remnant of the bilateral lesions that were removed. The postoperative mortality, therefore, is zero, and the case mortality is 5.5 per cent.

It is well known, however, that mortality per se does not tell the whole story, and, therefore, it is of interest that 15 of the patients are back at their usual occupations, while 2 are still sufficiently upset mentally to require institutional care. The remaining patient was the 1 already mentioned, whose death occurred about 6 months after leaving the hospital.

DISCUSSION

There are but two or three points that need special emphasis. In the first place, bilateral hematomas, in this series, as in others were fairly common, there being 5 in 18 patients, or 27.7 per cent. This is a point that has been stressed by many writers on the subject and indicates again how necessary it is to make taps over both cerebral hemispheres, even though clinical signs point to a unilateral lesion.

Equally important is our firm conviction that chronic subdural hematomas should always, if possible, be treated by the simple method of tapping described above rather than by the more elaborate, and in our opinion more dangerous, bone flap operation. We cannot agree with Furlow that the latter is the treatment of choice, even in patients that are conscious and in good general condition, because the facts do not bear out this conclusion. The mortality figures that

are given by McKenzie[6] and by Fleming and Jones[7] of cases in which simple taps were used are 9 per cent and 12 per cent, respectively, while among the cases forming the basis of the present paper there were no operative deaths. Fourteen of our patients were in good general condition while 4 were comatose. It is true that in Gardner's[8] series of 20 operative cases, 12 patients were treated by craniotomy with no deaths resulting, whereas 2 died of the remaining 8 whose hematomas were evacuated by simple trephine openings. Gardner[8] himself says, however, that the type of procedure in these two instances was not responsible for their deaths, inasmuch as both were practically moribund at the time they were operated upon.

The only other recent article that advocates a more elaborate type of operation than tapping is that by Grant.[9] Among his 15 patients that were operated upon, 7 were subjected either to an osteoplastic flap or to a subtemporal decompression with no deaths resulting, while of 8 that had unilateral or bilateral burr openings, 4 died. From these figures alone, it would appear that a large craniotomy is preferable to the simpler procedure, but of the 4 deaths, 2 occurred because the burr openings were not made over the hemisphere where the clot was situated, and the lesion, therefore, was not treated but was found on the opposite side at autopsy. A third patient died of lung abscess and pneumonia, which might have occurred no matter what procedure was used. In the fourth case, Grant[9] himself states that "the clot was more solid than fluid", and in this instance a bone flap would doubtless have been the procedure of choice. In contrast to the figures quoted from McKenzie,[6] Fleming and Jones[7] and those of our own series, when osteoplastic craniotomy is employed routinely, as in Furlow's series, the operative mortality as given by him was 28.5 per cent. When hematomas are fairly well organized, however, there is no recourse but to evacuate them by turning down a bone flap. This was necessary in 4 out of the 18 cases in our series.

CONCLUSIONS

1. Chronic subdural hematoma is a clinical entity that must be differentiated from brain tumor, cerebral arteriosclerosis and thrombosis, postencephalitis syndromes and persistent headaches of unknown origin.

2. The diagnosis of the condition as well as the evacuation of the clot can be accomplished in most instances by simple small trephine openings over either side of the skull.

3. The prognosis both for life and for future usefulness is excellent, and the operative mortality, while varying with the state of the patient, should probably be less than 10 per cent.

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FEMORAL HERNIA

BY FRANKLIN G. BALCH, JR., M.D.*

DEFINITION. A femoral hernia is a protrusion of abdominal viscera through the femoral ring into the femoral canal.

Anatomy. The mouth of the femoral canal is bounded anteriorly by the inguinal (Poupart's) ligament, posteriorly by the origin of the pectineus muscle from the pubis, medially by the lacunar (Gimbernat's) ligament, and laterally by the femoral vein. A femoral hernia usually descends between the lacunar ligament and the femoral vein.

Sac. The sac is a protrusion of the peritoneum through the femoral ring. Its content is usually the omentum.

Complications. Foss and Hicken[8] report that 33 per cent of 60 cases of femoral hernias were strangulated when admitted to the hospital. Erdman[7] states that recent reports from the New York and Mount Sinai Hospitals in New York City show, respectively, 26 and 32 per cent strangulations in all cases admitted with the diagnosis of femoral hernia. In our series of 100 cases at the Massachusetts General Hospital from 1924 through 1933, there were 9 cases of strangulation. In 14 additional cases with incarceration of the omentum, the bowel was not involved. Of the 9 cases of strangulation, 3 were of the Richter type in which only a portion of the bowel was strangulated. In inguinal hernia the incidence of strangulation is said to be from 2 to 4 per cent.

Almost all of the abdominal viscera, with the exception of the spleen and liver, have been found in femoral hernias. In 1 case[15] of this series the contents of the sac proved to be a Meckel's diverticulum. In another, about 10 inches of the transverse colon had prolapsed

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through the ring but did not require resection. In another hernia the sac contained the cecum, most of the ascending colon, several feet of terminal ileum, and most of the omentum. A portion of the omentum, the size of a football, was removed, the patient surviving the operation but dying about 1 month later, apparently of heart disease. Resection of the bowel was necessary only once, 3 inches of the small intestine being removed. This patient died 18 hours afterward of arteriosclerotic heart disease. The ages of these cases were 72 and 67 years, respectively.

Side Involved. Most writers state that the right side is more frequently involved than the left. This is in accord with our findings that 65 hernias were on the right and 27 on the left. Eight patients had bilateral hernias. Foss and Hicken[8] report 2 bilateral cases out of 60, and DeGarmo[6] 11 cases out of 99.

Sex. Foss and Hicken[8] state that the ratio of males with femoral hernias to females was 35 to 65. Erdman[7] concludes that it may vary anywhere from 1 to 2, to 1 to 4. DeGarmo[6] reports 16 males and 83 females. Shawan and Altman[14] found that of 97 cases, 43 were in men and 54 were in women. At the Massachusetts General Hospital there were 48 cases in males and 52 in females (table 1).

TABLE 1

	No. of Cases	Side			Sex Ratio		Percentage of Strangulations
		Right	Left	Bilat.	Male	Female	
Foss and Hicken	60	More often	—	2	1	1.9	33
Erdman	—	—	—	—	1	3	26—N. Y. Hosp. 32—Mt. Sinai
DeGarmo	99	—	—	11	1	5+	—
M. G. H.	100	65	27	8	1	1+	9—(3 Richter)

Age. Femoral hernia is seldom encountered in children, and Watson[16] states that it rarely occurs before the twentieth year. DeGarmo[6] reports 4 cases under 10 years of age and 6 more between the ages of 10 and 20. B. L. Coley[3] had 14 cases in children of which 4 were in males and 10 in females. In the Massachusetts General Hospital series 3 cases occurred under the age of 10, and 9 between the ages of 11 and 20. Table 2 shows the age distribution. It will be noted that the largest number of cases occurred between 30 and 60 years of age.

Signs and Symptoms. The symptoms are usually mild or absent except in the presence of strangulation or when omentum adherent within the sac causes a pulling sensation in the epigastrium. The femoral hernia usually manifests itself as a small swelling below the inguinal ligament, but it is occasionally very difficult to differentiate from an inguinal hernia. If the surgeon is in doubt, he is probably dealing with the femoral variety; occasionally a psoas abscess, femoral adenitis, or a lipoma may be mistaken for a femoral hernia.

Treatment. All surgeons condemn the use of a truss except in very rare cases. Keynes,[9] surgeon to the City of London Truss Society, states that "femoral herniae should never be treated by a truss unless the patient refuses operation or is obviously unfit for an operation even with local anesthesia." DeGarmo[6] of the New York Post-Graduate Medical School and Hospital states he has never seen a cure resulting from the use of a truss. Mechanically a truss cannot be kept in place.

In general there are two methods of operative approach, the femoral and the inguinal. Numerous technical departures from the classical methods of closing the femoral canal have been developed by and named after various surgeons, but the underlying principle has been the same in all. There has even been some discussion as to whether it is necessary to obliterate the canal. Ochsner[12] wrote in 1906, "It is a well-known fact that it is practically impossible to keep a circular opening in any part of the body from closing spontaneously unless it be lined with a mucous or serous membrane. The anatomic relations of the femoral canal are such as to form ordinarily a definite circular opening." He reported no recurrences in 30 cases treated by removal of the sac without closure of the canal. Keynes,[9] on the other hand, claimed in 1927 that "removal of the sac is not curative." He reported that in "10 patients the hernia was treated by laparotomy with complete removal of the sac from within the peritoneal cavity. In 6 of the cases so treated, the hernia promptly recurred." He has abandoned the "low operation" (femoral approach) and advocates the "high operation" (inguinal approach), which he feels should always be employed in cases of strangulation. Pott, quoted by Carscadden,[2] reported that 36.7 per cent of hernias recurred when the sac only was removed, and that 23.6 per cent recurred when the ring was closed in addition. Bresset's figures, as quoted by the same author, are 29 per cent, and 8.6 per cent, respectively. He did not state the method of approach. Gask, also quoted by Carscadden, reported removal of the sac through a laparotomy wound in 10 cases with recurrence in 6. Counseller and Cox[5] in 1933 advised the use of the femoral approach through a vertical incision. They close the canal by sewing the pectineus fascia and Gimbernat's ligament to Poupart's ligament, using chromic catgut as their suture material. In the classical femoral approach some form of purse-string suture has been used by Cushing and Coley. Bassini, DeGarmo, Ferguson and others have employed

other methods of suture. At the present time most surgeons feel that the canal should be obliterated after removal of the sac.

We feel that the method to be employed depends on several factors. The femoral route is usually the simpler, giving good results in the smaller uncomplicated cases. The inguinal route should be employed in the larger hernias, in those combined with inguinal hernia on the same side, and in all cases of strangulation. In certain other instances, the combined approach may be indicated, cutting the inguinal ligament if necessary.

Operative Accidents. Injury to the bladder is the most frequently encountered accident, especially when the inguinal approach is used. Great care must be taken not to injure the deep epigastric vessels or the femoral vein. This is particularly easy to do in cases of obesity. Watson[16] reports a series of such accidents. In the series at the Massachusetts General Hospital the bladder was opened in 1 case where the combined inguinal and femoral operation was employed, and in 2 other cases the epigastric vessels were punctured. All 3 patients made uncomplicated recoveries.

Anesthesia. In the Massachusetts General Hospital series, ether was employed in 60 per cent of the cases, local anesthesia in 21 per cent, spinal anesthesia in 16 per cent, gas and oxygen in 2 per cent, and avertin in 1 per cent. Recently, spinal anesthesia in doses of about 150 mg. of novocain has been the method of choice. Watson[16] advocates local anesthesia in all but young children.

Mortality. There were but 2 deaths in the series of cases from the Massachusetts General Hospital, both of which have been mentioned above. This makes an operative mortality of 2 per cent. As 1 of these deaths occurred 1 month after operation, however, it is, perhaps, not fair to include it in operative mortality.

Postoperative Care. This is essentially the same as that for inguinal hernias. The patient should be kept in bed from 12 to 14 days.

Recurrence. Resekke, quoted by Watson,[16] reports that in a series of 170 patients examined "several years after operation by the femoral route in which the opening was closed by a mattress suture, there were 35 per cent of recurrences." Watson[16] also states that "DeLuca reported 57 operations by the femoral route with no recurrences." Bassini, quoted by W. B. Coley,[4] claimed that there were no recurrences in 41 cases operated upon by the femoral approach. B. L. Coley[3] in 1924 "found 10.7 per cent recurrences in 28 cases re-examined following the femoral operation." Erdman[7] states that "in our own experience with the inguinal operation, recurrences reached 17 per cent, including one direct hernia which evidently was due to the trauma in the inguinal canal." Keynes[9] reports that in a series of 365 recurrent hernias collected from 1917 to 1926, 90 were femoral. He does not state by what method the operation was performed or what was the total number of femoral hernias operated upon.

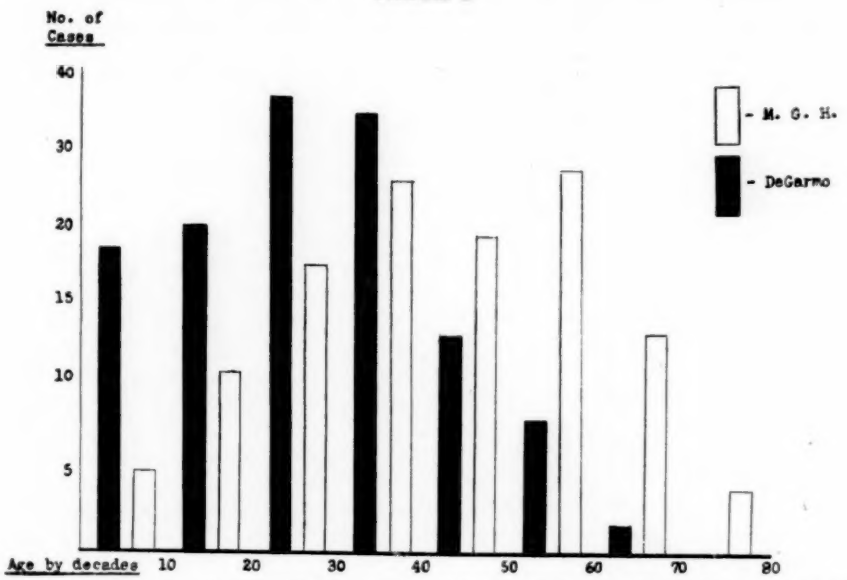
The following table compiled by Watson[16] shows the combined results from several clinics:

TABLE 3

Method	No. Cases	No. Recurrences	Percentage of Recurrences
Femoral operation with closure of ring	469	19	4.05
Suture of Cooper's to Poupart's ligament	769	33	4.23
Muscle plastic operation (Galyer)	182	18	9.89
Operation by the inguinal route	206	10	4.85

In the Massachusetts General Hospital series, 62 patients were examined 1 year or more after

TABLE 2



operation. Of these, 10, or 16.2 per cent, recurred. Six of these had been operated upon by the femoral approach and 1 by the inguinal. Of the entire group of 62 cases, 43 had been closed by the femoral route, 5 by the inguinal and 4 by the combined. This gives a recurrence rate of 14 per cent by the femoral route, and 20 per cent by the inguinal. Obviously these statistics are of little value because of the few cases operated upon by the inguinal route.

In 8 cases it was necessary to cut and resuture Poupart's ligament. In 4 of these there was no recurrence. Of the remaining 4, the hernia recurred in 2, while the other 2 patients could not be traced. The use of fascia from the external oblique muscle or fascia lata apparently prevented recurrence in 4 patients, in all of whom the hernia had been closed by the inguinal route. Carscadden[2] in 1934, using the inguinal approach, reported 6 similarly treated cases with no recurrence. He advocated the use of a special needle holder with which to pass the fascia.

TABLE 4

FEMORAL HERNIA—RECURRENCE

	Closure of Sac Only		Closure by Ing. Approach		Closure by Femoral Approach	
	No. of Cases	Percentage of Recur.	No. of Cases	Percentage of Recur.	No. of Cases	Percentage of Recur.
Ochsner	30	0	.....	.....	.....	.....
Keynes	10	60	.....	.....	.....	.....
Resekke	.....	.....	.....	.....	170	35
Pott	933	36.7	.....	.....	.....	.....
Bresset	Not stated	29	.....	.....	.....	.....
Gask	10	60	.....	.....	.....	.....
DeLuca	.....	.....	.....	.....	57	0
Watson	(Combined group)		206	4.85	469	4.05
B. L. Coley	.....	.....	.....	.....	28	10.7
W. B. Coley	.....	.....	.....	.....	69	0
Erdman	.....	.....	Not stated	17	.....	.....
M. G. H. series	.....	.....	5	20	43	14
Foss and Hicken	Report no recurrences out of 60 cases. No mention made of method of closure.					
Bassini	.....	.....	.....	.....	41	0
DeGarmo	.....	.....	.....	.....	110	.9+

CONCLUSIONS

Based on an analysis of 100 cases of femoral hernia as seen at the Massachusetts General Hospital between 1924 and 1933, and a short review of the appropriate literature, we believe that:

1. Femoral hernia occurs as frequently in men as it does in women.
2. Palliative treatment with a truss is inadequate and even dangerous.
3. The inguinal approach should be used in operating upon all large femoral hernias, all femoral hernias that are combined with inguinal hernias on the same side, and all strangulated femoral hernias.
4. If the femoral approach is used, it may be necessary to cut Poupart's ligament. This appears to increase the risk of recurrence.
5. The recurrence rate by either method of approach lies between 10 and 20 per cent.
6. The use of fascia from the external oblique muscle or fascia lata, though making the operation more difficult, probably lessens the chance of recurrence. It is most useful in dealing with the larger recurrent hernias.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23091

PRESENTATION OF CASE

A 70 year old American widow entered complaining of intermittent abdominal pain.

The patient stated that she had always been troubled mildly with indigestion, gaseous eructations and gas pains, but had remained relatively well until 2 years before entry. At that time she had an attack of dysuria, urinary frequency and fever, lasting for about 12 days. She had had several minor attacks of cystitis previous to that time. During the few months following her severe attack there was an occasional recurrence of the dysuria and frequency. Fifteen months prior to admission she developed severe cramplike pains in the left lower quadrant, associated with diarrhea and bloody urine. This developed into an acute pelvic peritonitis that localized in about 10 days to a palpable, tender mass in the left lower quadrant. A laparotomy was then performed at another hospital, and an abscess containing about 500 cubic centimeters of pus was evacuated. Two months later the wound had entirely healed, and she was apparently well.

During the year preceding admission to this hospital she had occasional cramplike pains in the midabdomen, but had no other significant complaint except for moderate anginal pain on effort. One month preceding admission there was sudden recurrence of dysuria and frequency, which had continued. Two weeks before entry a small abscess behind the cervix was incised and drained. It contained only about 30 cubic centimeters of pus, but discharged copious amounts of foul-smelling pus, suggesting fecal material, for the succeeding 2 weeks. Following drainage of the abscess the midabdominal cramps increased in frequency and severity, and occasionally woke her at night. There was occasional nausea, and she vomited once a few days before admission. The vomitus was watery in character. Her bowel habits were unchanged. There was moderate anorexia. During the last few months of her illness her weight increased from 162 to 169 pounds.

Physical examination showed a well-developed, slightly obese woman in no acute discomfort. The thyroid was just palpable. The heart was slightly enlarged to the left, and there was a blowing systolic murmur at the apex. The blood pressure was 130/80. The lungs were clear. The liver edge was palpable 4 centimeters below the right costal margin. There was a firm, rounded, slightly tender mass situated in the midline, halfway between the umbilicus and the ensiform. This mass was not movable and did not change position on deep inspiration. It appeared to be about the size of a small grapefruit. There was no pulsation or audible bruit. There was moderate, pitting edema of both legs. The knee and ankle jerks could not be obtained.

The temperature, pulse and respirations were normal.

Examination of the urine showed a specific gravity of 1.006. There was no albumin or sugar, and the sediment contained an occasional white blood cell but was otherwise negative. The blood showed a red cell count of 4,600,000, with a hemoglobin of 80 per cent. The white cell count was 9,200, 74 per cent polymorphonuclears. Repeated stool examinations gave negative reactions to the guaiac test. The nonprotein nitrogen of the blood was 17 milligrams per cent. The serum protein was 5.3 grams per cent and the blood chlorides were equivalent to 102 cubic centimeters of N/10 sodium chloride. The CO_2 combining power was 63 volumes per cent. A fasting blood sugar was 106 milligrams per cent.

A Graham test showed a normal gallbladder. A gastrointestinal series demonstrated a normal esophagus and a stomach that was smooth in outline. The antrum was displaced upward by the abdominal mass, which was entirely within a markedly widened duodenal loop. The mucosa of the stomach and duodenum showed no evident abnormality. A barium enema exhibited numerous diverticula and moderate spasm in the descending colon, but the sigmoid and rectum were smooth and showed no diverticula. There was no evidence of obstruction. An intravenous pyelogram showed displacement of the left kidney outward, but not downward. There was no deformity of the pelvis and no evidence of block in either ureter.

On the sixth hospital day an exploratory laparotomy was performed.

X-RAY INTERPRETATION

DR. GEORGE W. HOLMES: This patient was examined at the Phillips House and then reexamined at the Baker. There is an interval of a few days. About all that I can see to point out in these films taken at the Baker is that the gastrointestinal tract is essentially negative except for displacement of the gall bladder. Here is the film that shows best the mass within the duodenal loop. It is a very large mass. It lies apparently within the loop and displaces the stomach downward and toward the left. A malignant tumor of that size in the head of the pancreas would in all proba-

bility infiltrate the wall of the duodenum sufficiently to cause some obstruction or some change in position. This is a perfectly normal film without evidence of obstruction. A cystic tumor of the head of the pancreas might do that but I hardly think a malignant tumor could. There is nothing interesting in the colon.

DR. AUBREY O. HAMPTON: The clinical diagnosis at one exploration was diverticulitis of the sigmoid. They thought that the abscess was due to displacement of the sigmoid, and I was surprised to find that it was normal in that area.

DR. HOLMES: This is the examination of the genitourinary tract by the intravenous method. You can see that the left kidney is displaced outward, not downward. Its pelvis is not distorted. There is no evidence of block in either ureter. The kidney must be rotated and displaced by the mass. In this film you can see the opposite kidney quite well, and it appears normal. We have a large, round, smooth retroperitoneal mass surrounded by the duodenal loop which is displacing the kidney outward and rotating it.

DIFFERENTIAL DIAGNOSIS

DR. ARTHUR W. ALLEN: There are a great many interesting features about this case, but I think it is a very difficult one to tie up with the symptoms that she has had. The long period of indigestion would suggest that perhaps this lesion sprang from the gastrointestinal tract or from her pancreas originally, but a great many people have some indigestion all their lives and possibly this lead may not be too important. Another interesting factor is the onset of urinary symptoms coincident with pelvic inflammation, and pelvic abscess two years prior to entry. This might point toward either the lower gastrointestinal tract, that is, an abscess which developed from a diverticulitis, or it might originate in a lesion of the pelvic organs themselves. A woman of this age may have malignancy on which an abscess could develop and secondarily cause urinary disorder. Of course, one ought to consider very seriously some primary involvement of the urinary tract. Her story of bloody urine might not be an accurate record. Possibly the blood came from the vagina; so, unless we were told that this urine was a catheter specimen, we would not know whether the statement of bloody urine was important. At any rate, it was 2 years before entry, and when she came in here, a catheter specimen showed no blood at all. The origin of the pelvic abscess interests me because, if it were based on malignancy and was incised and drained, the chance of its healing spontaneously without a fistula would be pretty slight. That is true also of diverticulitis that forms an abscess. If you drain such an abscess a persistent fistula, however small, that will occasionally allow a little gas to escape, and once in a while a little fecal matter, is a very common occurrence. It is not common to have an abscess develop on a diverticulitis and heal tightly within 2 months, as this abscess did, but it would be possible to explain such a situation on the basis of pelvic inflammation.

The respite that this woman had between the attacks would seem to indicate that the fulminating process which she had 2 years before entry, with the abscess formation that was drained successfully and healed, may not in any way be connected with the present illness. I think we must bear in mind that we might be able to discard that 2 year old story in trying to decide what is going on now.

There is one thing about this patient that we would like to know and that is what the pelvic examination showed or what the rectal examination showed, as far as the pelvic viscera were concerned. It is not noted here and perhaps we should not ask, but that would be of considerable help in trying to decide whether the pelvis had pretty well cleared up before this admission. The facts that she had had a small abscess drained behind the cervix 2 weeks prior to admission and that there was a prolonged discharge of foul-smelling material which was fecal in odor would make one suspect that there had been a process going on in the pelvis since her episode of 2 years before. The fact that only 30 cubic centimeters of pus escaped at the time the abscess was opened does not throw much light on the size of the cavity, as it might simply have opened into one pocket and then other pockets or a larger cavity drained spontaneously into the smaller one as time went on. It might have contained fecal material, but I doubt it, because the record states that at this admission there had been no change in bowel habit. Usually when you have a fistulous tract in the intestine there is a definite influence over bowel habit, particularly if it springs from an inflammatory lesion in the colon, and there is apt to be a complete cessation of bowel movements for a while, even though discharge from the fistulous tract may be inadequate.

Her cramplike pains suggest intestinal disease of some sort but they are not well described. They do not tell us much other than that they were from the intestinal tract. They probably did not arise from the colon, as they localized in such a way that one would have to suppose that the lesion was interfering with the small bowel. There is no note of what the peristalsis sounded like through the stethoscope, and it might have been helpful to know.

Her apparent gain in weight I think might be explained on the basis of edema rather than on her state of nutrition. The x-ray examination is helpful in some respects and disappointing in others. Of the various conditions that could cause a mass of this sort one would think first of some kind of tumor arising in the pan-

creas. Dr. Holmes has pointed out from the x-ray examination that such was not likely, but one could get extension of a pancreatic tumor that might fill the area that we see in the x-ray plate, particularly if it was cystic. We are sure of one thing about the tumor; it is retroperitoneal, whether it be primary there or secondary. It is firmly fixed to the posterior parietal abdominal wall, it is difficult to move, and it does not rise and fall with respiration. So we have either a tumor arising from the pancreas or from the kidney or from the adrenal, or it is in the retroperitoneal glands. The record states very carefully that it does not pulsate, so I assume we can rule out aneurysm. I would have a difficult time trying to connect this tumor with the trouble in the urinary tract unless we could visualize this as being a hypernephroma of this kidney, and I have never known one to take this distribution. Such a tumor usually extends into the flank, but I suppose it could assume this position.

I am unable to make a definite diagnosis. I would limit my diagnosis to a retroperitoneal tumor of unknown origin and expect that we might find, if supported by the pelvic examination, that it was a continuous process with whatever was going on in the pelvis and that it might be malignant glands from this area, a lymphoma, a pancreatic cyst or, possibly, a hypernephroma in an odd position.

Dr. Tracy B. Mallory: Are there any other suggestions?

Dr. Harrison E. Kennard: At operation a transverse incision was made and a mass was felt inside of the loop of the duodenum, which was about the size of a half a grapefruit. An incision was made through the gastrocolic omentum into the lesser peritoneal cavity, and the mass could be felt behind that. Then, after lifting up the transverse colon, an incision was made through the transverse mesocolon to obtain better exposure. The mass felt round and soft, but definitely noncystic. A biopsy was taken. There were a few adhesions to be felt in the pelvis, but a thorough examination was not possible on account of the two previous operations. The preoperative diagnosis was question of cyst of the pancreas, and the postoperative diagnosis was retroperitoneal malignancy, question of lymphoblastoma. The postoperative course was excellent for the first few weeks. The wound healed by first intention, and she got out of bed on the thirteenth day. Deep x-ray therapy was started on the twelfth day but precipitated severe vomiting. She had two deep x-ray treatments, and after the second one it was thought unwise to continue because she vomited so much. She stopped vomiting after about 3 or 4 days and from then on improved slowly for several weeks and stayed in the hospital only because she wanted to be entirely well before going on a long trip. After about 6 weeks she went downhill again and vomited on several occasions. She then turned sharply for the worse, and lost her appetite. Her bowels became constipated, and she was able to move them only by the use of frequent catharties. She finally developed bronchopneumonia and died 10 weeks after the last operation.

There was one other point—edema of the lower legs appeared. It was present when she first came into the hospital but became much more marked just before she died.

Clinical Diagnosis

Pancreatic cyst.

Dr. Arthur W. Allen's Diagnoses

Retroperitoneal tumor:

1. Metastatic malignant glands?
2. Lymphoma?
3. Pancreatic cyst?
4. Hypernephroma?

Anatomic Diagnoses

Retroperitoneal lymphosarcoma.
Thrombosis of the inferior vena cava and the common iliac veins.
Pelvic peritonitis, chronic fibrous.
Diverticula of the colon.
Bronchopneumonia.

Pathological Discussion

Dr. Mallory: The autopsy showed a very large tumor mass in the retroperitoneal tissue which completely encircled the aorta and the vena cava. The vena cava was invaded by the tumor, and its wall was extensively replaced by tumor tissue, but it was not obliterated by tumor growth. Below the point where the tumor involved it, however, it had become thrombosed, and the terminal 5 centimeters of the vena cava and both the iliac arteries were completely filled with adherent blood clot. I believe that was a terminal event of only a few days' duration. The mass also occluded the left ovarian vein, but the right ovarian vein was free so that there was a possibility of a little collateral circulation developing. Both renal arteries were completely surrounded by tumor but neither of them was occluded. Considering the extent of the tumor mass and the number of organs involved, it is rather surprising that the symptoms were not more pronounced than they were. The pelvis contained a mass of inflammatory adhesions tying the tubes and ovaries into a single large ball, which was also adherent to the lower sigmoid. We found numerous diverticula in the descending colon and upper part of the sigmoid, but in the region of the inflammatory adhesions no diverticula were found. So we cannot say with certainty what the cause of the pelvic inflammation was. I think it was prob-

ably a true pelvic inflammatory disease rather than the results of a ruptured diverticulum.

A PHYSICIAN: Was the abscess that she had 15 weeks before caused by a diverticulum?

DR. MALLORY: I do not think so.

A PHYSICIAN: Where did the tumor arise?

DR. MALLORY: It was a lymphosarcoma evidently starting in the retroperitoneal nodes.

A PHYSICIAN: Was the pancreas infiltrated?

DR. MALLORY: No, it was displaced but was entirely negative.

CASE 23092

PRESENTATION OF CASE

A 54 year old native, female schoolteacher was admitted complaining of dyspnea.

About 9 months before entry, following an emotional upset, the patient noted the rather abrupt onset of dyspnea. Questions relative to her mental distress caused the patient to choke and cry, but she was unable to give any details. Associated with the dyspnea there was complete loss of appetite, but she ate sugar in order to sustain herself. Her strength and weight diminished so that after 2 months she had lost 20 pounds. During this time she was unable to sleep and became very irritable. The dyspnea progressed, and she developed a dry, hacking cough. Six months before coming to the hospital she noticed a craving for sour foods, such as grapefruit, and further questioning revealed the fact that she had craving for all foods except those that were sweet and sugary. Coughing spells occasionally caused her to gag and to vomit the food she had eaten. The cough was particularly bad when she lay on her back or on her right side and frequently awakened her at night, although there was no associated dyspnea or wheezing at that time. On the day of entry she coughed up a small amount of red, frothy material for the first time. During the 2 months preceding entry she had regained 15 pounds.

Physical examination showed a thin, flushed, slightly cyanotic, sick-looking woman propped up in bed and lying on her left side. There was marked respiratory discomfort. There was clubbing of the fingers. The mucous membranes were pallid. The chest was thin and expansion was diminished on the left side. There was dulness to flatness in the left chest extending posteriorly from the base to a point just above the angle of the scapula, and anteriorly up to the fifth rib. The breath sounds in this region were decreased in intensity, tactile fremitus was absent, but egophony was elicited. Just above the area of dulness, moist rales were audible. Percussion of the heart was inconclusive because of dulness in the left chest. The impulse was felt in the sixth interspace, 7.5 centimeters to the left of the midsternal line. An occasional extrasystole was noted, and there was a systolic murmur, transmitted into the neck vessels, best heard in the second right interspace. There was also a systolic thrill. The second aortic sound was absent. The radial pulse was 100 and was plateau in character. The blood pressure was 115/70. Palpation of the abdomen demonstrated a large, movable, hard, nodular, nontender mass in the right flank, extending anteriorly almost to the umbilicus. The spleen extended four fingers beneath the costal margin. Rectal examination revealed the presence of external and internal hemorrhoids.

The temperature was 101°F. The respirations were 30.

Examination of the blood showed a red cell count of 3,600,000, with a hemoglobin of 50 per cent. The white cell count was 5,900.

Several hours after entry the patient became rather markedly dyspneic but was partially relieved by an opiate. Shortly afterward she suddenly developed what appeared to be acute pulmonary edema, became markedly cyanotic and pulseless, and died within a short time.

DIFFERENTIAL DIAGNOSIS

DR. SYLVESTER MCGINN: In reviewing this patient's history it appears that she was suffering from left ventricular failure of 9 months' duration, characterized by shortness of breath and inability to lie flat at night because she was awakened by coughing spells. Not infrequently we see patients complaining of waking at night with cough rather than the more common type of cardiac asthma characterized by extreme breathlessness. This is apparently the result of the fact that the pulmonary congestion has not proceeded far enough to cause actual pulmonary edema but, nevertheless, produces a cough that wakes them. They then assume an upright position and thereby abort the attack.

The expectoration of red, frothy material is consistent with early left ventricular failure. I believe the absence of a story of peripheral edema forces one to assume that only the left ventricle was involved at that time.

Her other symptoms consist of gastric disturbances that may well have been caused by digitalis intoxication, although we have no definite information in regard to the use of that drug. Weight loss could result, if this supposition were correct, and weight could later be regained, owing to congestive failure. In the treatment of cardiac patients, moreover, it is not uncommon to find that they have difficulty in eating, even without digitalis.

On entry to the hospital she apparently was much sicker than she had previously been, and it would seem that she died a cardiac death. In the physical examination the most definite part of the picture appears to be caused by aortic stenosis, characterized by a systolic murmur heard in the second right interspace, trans-

mitted to the neck, associated with a thrill and the absence of the aortic second sound. This can mean but one thing when the thrill is present—stenosis of the aortic valve. Other findings in the physical examination are not so clear. The left chest is known to have dulness on percussion. I assume that the auscultatory sounds were caused by fluid over compressed lung and to an enlarged heart.

Another possibility that must be considered is metastatic malignancy. Her age is in favor of such a diagnosis, and two masses have been felt in the abdomen, though one of them was apparently thought to be the spleen. We are reminded of her distaste for food, since that is a common symptom in cancer of the stomach. Her secondary anemia is also consistent. The large nontender mass in the right flank, extending to the umbilicus, may be the description of an enlarged liver. Other possibilities for this mass are hydronephrosis, hypernephroma and malignancy of the bowel.

Clubbing of the fingers was noted. This condition makes one think of a congenital heart, but it is usually associated with cyanosis or at least with some defect in the cardiovascular system allowing the admixture of venous and arterial blood. It could come from chronic pulmonary disease and in this case might be caused by long-standing metastases in the lungs. It could come from subacute bacterial endocarditis, and that diagnosis would account for the anemia and the enlargement of the spleen. However, there is no other information, such as a septic temperature or the presence of petechial hemorrhages, which would allow one to consider this diagnosis further. Finally, some people naturally have clubbed fingers without any known organic disease to explain them. The white cell count seems to be within normal limits, and I feel that this rules out the necessity of considering various blood disorders.

There are many other things that we would like to know about in this case, especially what an x-ray of the chest would show, and further information in regard to the urinary findings and the abdomen, whether ascites or edema was present.

My diagnosis is left ventricular failure, the result of aortic stenosis, probably owing to calcareous valve changes, inasmuch as there is no rheumatic history. Although one third of the cases of aortic stenosis are associated with mitral stenosis, there is no evidence that this lesion exists in the present case.

Clinical Diagnoses

Aortic stenosis.
Pulmonary edema.
Renal malignancy.
Splenomegaly.
Secondary anemia.

Dr. Sylvester McGinn's Diagnoses

Aortic stenosis, with left ventricular failure.
Hydrothorax.
Congestion of the liver and the spleen.

Anatomic Diagnoses

Aortic stenosis, calcareous.
Acute rheumatic myocarditis.
Subacute bacterial endocarditis.
Cardiac hypertrophy.
Hydrothorax, bilateral.
Ascites, slight.
Hydropericardium.
Pulmonary edema and congestion.
Splenomegaly.
Pulmonary osteoarthropathy.
Cholecystitis, chronic.
Hydrops of the gall bladder, marked.
Cholelithiasis.
Leiomyomata uteri, with calcification.
Cortical adenomata of the adrenal.

Pathological Discussion

Dr. Tracy B. Mallory: This patient's dramatically sudden death prevented completion of various further studies that had been planned. The doctors on service agreed with Dr. McGinn that she certainly had aortic stenosis with congestive failure and paroxysmal pulmonary edema. They thought the mass in the right half of the abdomen was a malignant tumor of the kidney, and they noted the splenomegaly without attempting to explain it. The autopsy showed that the mass was of no importance in relation to her death. It consisted of an immensely dilated gall bladder, 16 centimeters in length, resulting from an impacted stone at the mouth of the cystic duct and a secondary hydrops of the gall bladder.

The heart showed, as everyone expected, a marked aortic stenosis. This was of the typical variety seen in elderly people, which is characterized by extensive calcareous deposits and is not accompanied by any abnormality of the mitral valve. As is so often the case in these calcareous aortic lesions, one commissure showed a much more marked degree of fusion than the other two and for that reason strongly suggested as an underlying basis a congenital bicuspid aortic valve. This was by no means the entire story in this case, however. Whereas the aortic surface of the cusps were covered with irregular, projecting nodules of calcified material, the ventricular surfaces showed minute, pinkish, fresh vegetations. The mitral valve showed a slight calcification near the base of its longer cusp but was entirely free from fresh vegetations or from any lesions of the chordae tendineae that would suggest either acute or chronic rheumatic involvement. Microscopic sections of the myocardium, which, ex-

cept for hypertrophy, had appeared grossly negative, provided the greatest surprise when we discovered that the perivascular tissues were riddled with Aschoff bodies. Numerous small foci of fresh myocardial degeneration and some fibrous scars of varying age pointed to an extensive myocarditis.

The spleen was markedly enlarged, weighing 650 grams. On microscopic examination there were marked congestion, marked lymphoid hyperplasia and an unusually diffuse infiltration of the entire pulp with great numbers of large monocytes. The kidneys weighed 250 grams and showed, when the capsules were stripped, many minute, bright red petechiae. Microscopic examination proved them to be the result of a so-called focal or embolic glomerulonephritis. The lungs, as was expected, showed a marked grade of acute pulmonary edema, and there were a bilateral hydrothorax, a hydropericardium and a slight degree of ascites. Postmortem blood cultures were negative.

This case, like many others, still remains a difficult problem in differential diagnosis even after the autopsy has been reported. We have evidence of a calcareous aortic stenosis, a lesion which we believe is very rarely rheumatic in origin. We have apparently positive proof of acute rheumatic infection at the time of death, but we have certain features which cannot be explained by either of these diagnoses or by a summation of the two. The clubbed fingers, the anemia, the splenomegaly and the focal glomerulonephritis are all extremely suggestive of a bacterial endocarditis, and none of them are adequately explained by any of the other autopsy findings. Against such a diagnosis are the negative postmortem blood culture and the appearance of the vegetations. We unfortunately did not try to demonstrate organisms in them by direct smear at the time of autopsy. That bacterial endocarditis and rheumatic infection are often coincident has been pointed out by Pappenheimer and von Glahn, who even go so far as to suggest that subacute bacterial endocarditis cannot develop in the absence of active rheumatic infection. I am inclined to record this case as confirmatory evidence of their views.

The sudden death of the patient was not commented upon either by the physicians in the wards or by Dr. McGinn. I assume they both considered it to be a sudden attack of so-called cardiac asthma, which is really acute pulmonary edema. That was correct enough but, nevertheless, I think it deserved comment since death in an attack of cardiac asthma is certainly unusual as far as I can judge from postmortem material. On the other hand sudden death in elderly people with acute rheumatic myocarditis is by no means uncommon. It is also rather characteristic that in this age group joint symptoms are completely absent and the microscope alone can make the diagnosis of rheumatic infection.

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FORWARD AIMS OF MEDICAL EDUCATION

DR. LANGLEY PORTER, dean of the University of California Medical School, writing in the *Bulletin of the Federation of State Medical Boards* of the United States for December, 1936, offers some interesting if not entirely new views on the present curriculum and the aims of medical education. According to this thesis, if certain proper aims are to be accomplished, the medical school must be prepared to train four groups of graduates.

"The first group consists of men whose temperaments and aptitudes fit them to replace the vanishing general practitioner; men able to administer all available medical services to individuals and to families. . . . Secondly, every medical school should be prepared to train a part of its student body to engage in public health activities. This group should consist of men whose temperaments and abilities attract them to this field—men able to understand how the social organization most effectively may be used to accomplish all those community health tasks that can best be done through collective effort.

"A third obligation that rests on the medical school concerns its duty to provide a limited number of well-trained specialists and to revise present conceptions about what constitutes proper and legitimate fields for specialization. Finally, it is of the utmost importance that the need for highly trained investigators and teachers be remembered. No obligation of more urgency than this is imposed on those whose duty it is to guide students of medicine. No student body will develop more than a few such men. There will always be some blessed with brains, curiosity, pertinacity and patience, who will reward the school's interest in them."

These interests, and the interests of medical students in general, are not best served by dishing up a curriculum in watertight compartments, like a blue plate luncheon, or like a progressive Caribbean meal where each dish is a separate course, unrelated to the others. This, too often, is the present system under which so many hours are allotted to a course under an instructor who has no interests other than his own subject.

Integration and coordination should be the new watchwords in medical education, if medical education is to progress and fulfill its proper obligations. The semester system and the mosaic pattern of independent courses should be abolished in favor of a plan in which the total hours available are treated as a single course—a course "which attempts to integrate all the sciences and arts utilizable in practice, and which aims to produce a total based on a pattern of finding motifs that will appear in the very beginning of premedical education and be carried through until the completion of the student intern year."

The major guiding motifs, according to Dr. Porter, would naturally be physiology and pathology, with psychology, economics and sociology as essential components. In such a plan the faculty must remain the high command, with a flexible general staff functioning as a curriculum committee and being made up of every contributing branch in medicine. Under such a scheme "both the patient, as an individual, and the art of medicine, as opposed to the pseudoscience that now goes by that name," will be rediscovered.

Others have long held similar ideas concerning medical curriculums — students themselves not the least important among these. Definite steps already have been taken, and perhaps it is time to make further moves in changing medical education from a static condition to a dynamic force.

SIR JOHN BLAND-SUTTON

THE death of Bland-Sutton, December 20, 1936, at the age of eighty-one, brings to a close the life of a man of wide interests. Trained in natural history, he became one of the leading surgeons of London, long associated with the Middlesex Hospital. His skill brought to him the presidency of the Royal College of Surgeons of England, and for many years he was an important member of its Council. A devoted follower of John Hunter, he spent many hours in the Hunterian Museum, to which he added an important collection of specimens. To the Middlesex Hospital Museum he gave his material illustrating disease of the gall bladder and bile ducts.[1] In this field of surgery he made important advances.

Besides his achievements in surgery, Bland-Sutton will be remembered for two other aspects of his life. For years he was a close friend of Rudyard Kipling, one of the very few intimate friends that Kipling had in later life. It is said that Kipling would do things for Bland-Sutton that he would not do for any other man. In 1908, Kipling gave an address[2] before the Medical School of Middlesex Hospital, presumably at Bland-Sutton's request, which has become a rare and choice item to the book-lover. In it, one will find many tributes to the medical profession, written by one of the masters of English literature. Only one may be quoted here: "If you fly a yellow flag over a center of population you can turn it into a desert. If you choose to fly a Red Cross flag over a desert you can turn it into a center of population towards which, as I have seen, men will crawl on hands and knees." Kipling also wrote the introduction, in quaint English, to Bland-Sutton's autobiography,[3] a book of great interest, written when the author was seventy-five.

Bland-Sutton, secondly, continued his researches in zoology all his life. For years he did all the postmortem examinations at the London Zoo, and his writings are full of references to the anatomy and pathology of animals. At a meeting of the Association of Surgeons of Great Britain and Ireland, in 1920, he gave an instructive and amusing address on "Science and Surgery."[4] He covered such topics as omphalitis, the lacteals, the glandular pantheon (pituitary disorders), avian clubs, testicles and feathers, and lamps and specialism. Two quotations will indicate his delightful style: "Lymphatics are the subtile channels by which infective material reaches the circulation for distribution — and mischief." In speaking of the pituitary gland: "It is strange that overgrowth of the most cunningly concealed glandular body in man is responsible for making its owner the most conspicuous person in an assembly." Similar material will be found in the "Hunterian Oration" for 1923. English medicine has lost a powerful and colorful figure by the death of Bland-Sutton.

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THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

SALTER, W. T. M.D. Harvard Medical School, 1925. Tutor, biochemical sciences, Harvard University. Associate physician, Collis P. Huntington Memorial Hospital. Associate in medicine, Peter Bent Brigham Hospital. Research fellow in biochemistry, Harvard Cancer Commission. Assistant professor, Harvard Medical School. Address: Harvard Medical School, Boston.

LERMAN, J. M.D. Harvard Medical School, 1927. Assistant in medicine, Harvard Medical School and Massachusetts General Hospital. Address: Harvard Medical School, Boston. Their subject is "The Genesis of Thyroid Protein." Page 371.

CHURCHILL, EDWARD D. M.D. Harvard Medical School, 1920. John Homans Professor of Surgery, Harvard Medical School. Chief of West Surgical Service, Massachusetts General Hospital. Address: Massachusetts General Hospital, Boston. His subject is "Principles of Parathyroid Surgery." Page 376.

HASTINGS, A. BAIRD. Ph.D. Columbia University, 1921. Formerly, chemist, United States Public Health Service; assistant, Rockefeller Institute for Medical Research; associate, Rockefeller Institute for Medical Research; professor of physiological chemistry, University of Chicago; professor of biochemistry, Lasker Foundation for Medical Research, University of Chicago; lecturer, University of Southern California; visiting professor, Peiping Union Medical College, Peiping, China. Now, Hamilton Kuhn professor of biological chemistry, Harvard Medical School. Address: Harvard Medical School, Boston. His subject is "Factors Governing Calcium Equilibria in the Body." Page 377.

HORRAX, GILBERT. M.D. Johns Hopkins University School of Medicine, 1913. D.Sc. Williams College (Hon.), 1936. Neurosurgeon, New England Deaconess and New England Baptist

Hospitals. In charge of Neurosurgical Service, Lahey Clinic. Address: 605 Commonwealth Avenue, Boston.

POPPEN, JAMES L. M.D. Rush Medical College, 1930. Neurosurgeon, New England Deaconess and New England Baptist Hospitals. Neurosurgical Service, Lahey Clinic. Address: 605 Commonwealth Avenue, Boston. Their subject is "The Frequency, Recognition and Treatment of Chronic Subdural Hematomas." Page 381.

BALCH, FRANKLIN G., JR. M.D. Harvard Medical School, 1923. F.A.C.S. Assistant surgeon, Massachusetts General Hospital. Surgeon, Faulkner Hospital. Assistant in surgery, Harvard Medical School. Address: 330 Dartmouth Street, Boston. His subject is "Femoral Hernia." Page 385.

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CASE HISTORIES

NO. 9. HYDATIDIFORM MOLE

Mrs. E. J., a white primipara, aged 27, was sent by her physician to the hospital on February 29, 1932. Her last menstruation had started November 11. For the past 7 weeks she had been bleeding, which she attributed to the sudden death of her brother. The flow was sufficient to necessitate a napkin night and day, but she had had no cramps and had passed no tissue. Her doctor reported that previous to admission she had had a blood pressure as high as 158 (systolic) and that a recent urine examination had shown albumin and blood. During this same time the patient had complained of frequent headaches, blurring of vision, occasional vomiting and some edema of the ankles and face.

The family history was unimportant. The past history revealed the usual children's diseases, and, in 1918, influenza. The patient had never had scarlet fever or diphtheria. There had been no operations or serious accidents. She had had no complaints referable to the special senses or to the cardiorespiratory, gastrointestinal or genitourinary systems previous to the present illness. Menstruation commenced at 12 years of age and was regular at 28 day intervals. The periods lasted 5 days, and she had no dysmenorrhea or leukorrhea.

Physical examination revealed a well-developed and fairly well-nourished woman. Her face was slightly puffy. The pupils reacted normally. The ears, nose and throat were all grossly normal. There was no thyroid enlargement. The heart and lungs were normal. The abdomen showed a tumor, evidently the uterus, which was the size of a 5 to 6 months' pregnancy. There was some tenderness in the lower abdomen, but no spasm. There was moderate edema of the feet and ankles. On vaginal examination the patient showed a nulliparous perineum. The mucosa was deeply cyanotic, and there was a moderate muco-epithelial discharge. A moderate amount of blood was issuing from the cervix, which was patulous. No fetal parts could be felt, and there were no irregularities in the uterus.

At the time of admission the temperature, pulse and respiration were 99.6°F., 100, and 20, respectively. The blood pressure was 184/106. The urine showed a large trace of albumin, a moderate number of white blood cells, a few red blood cells and no casts. The white blood cell count was 9,100, with a normal differential count; the red count was 3,780,000, and the hemoglobin 70 per cent (Tallqvist). The Wassermann and Kahn tests were negative. The blood chemistry showed 30 mg. per cent of nonprotein nitrogen, 97 mg. sugar and 3.4 mg. uric acid. An x-ray taken on the day of entrance showed no signs of a fetal skeleton, although an Aschheim-Zondek test taken the following day was positive.

The diagnosis of hydatidiform mole was made, and on March 5 the uterus was emptied. Large amounts of grape-like masses were removed together with what appeared to be old placental tissue. This material was examined, and the pathological diagnosis of "hydatidiform mole, acute inflammation" was returned. The report read, in part, "In the specimen received there is no evidence of malignancy or the formation of a chorio-epithelioma."

The patient made a good recovery. On the sixth day postpartum she passed a large amount of gelatinous material and had some bright flow This stopped with the use of oxytocics. At the time of discharge, on the eleventh day after operation, the lochia was scant, but the fundus was still palpable two fingers above the umbilicus. An Aschheim-Zondek test was ordered before discharge, but unfortunately there is no report of this test in the record.

The patient was seen again on April 18 in the postnatal clinic at which time she showed no abdominal tenderness, masses or spasm. On vaginal examination the uterus was normal in

*A series of selected case histories by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

size and position, firm and freely movable. There was no flow. After this the patient was not seen again at this hospital.

On July 7, 1933, she was admitted to another hospital with the following interim history. From the time of leaving the first hospital her periods were irregular until October, when, after being 2 weeks overdue, she flowed profusely for 2 weeks. She was normal in December. In January, 1933, she again was 2 weeks late and flowed profusely for 2 weeks. Her periods were normal for 2 months, but since April she has had dark staining daily, although she has also had what she considers periods at regular intervals.

For 2 weeks previous to admission she had had a cough with occasional blood-streaked sputum. Her appetite was poor and she had occasional nausea. All other history was negative.

On examination she was found to be poorly nourished. Heart, lungs and abdomen were reported normal. On vaginal examination the uterus felt somewhat enlarged and soft, but the cervix appeared negative. The hemoglobin was 60. An Aschheim-Zondek test was done and was reported strongly positive in dilutions higher than 1-100.

A diagnosis of chorio-epithelioma was made, and a dilatation and curettage for biopsy of the endometrium was performed. Frozen sections of the tissue were unsatisfactory. Many large clots with necrotic-looking tissue were removed, and it was decided to do nothing further until other sections of the tissue were examined. These revealed chorio-epithelioma.

Five days later, and the day the tissue diagnosis was reported, rales were found at the base of the right lung, both front and back. On the following day a chest plate showed "many rounded dense masses throughout both lung fields due to metastatic carcinoma."

From that time the patient lost ground steadily and died on July 28. At necropsy the anatomic diagnosis of chorio-epithelioma of the uterus was made. There were metastases to the lungs, brain, kidneys, liver, spleen and intestines. This diagnosis was confirmed by microscopic examination of the tissues.

COMMENT

The diagnosis of mole should have been made earlier in this case. It is strongly suggested, even if no cysts are passed, by the too rapid increase in the size of the uterus in the presence of repeated bleeding. It could have been settled before the patient was sent to the hospital by a quantitative Aschheim-Zondek test. A positive test in a dilution of 1-50 is suspicious of mole, while a positive in a dilution of 1-100 is considered definite, if the accompanying symptoms are confirmatory.

The technical dangers of emptying the pregnant uterus are well known. The danger of perforation is more marked in conditions, such as mole, where the uterine wall may be still further weakened by the pathologic process. There is always, then, the difficult decision between trying to remove the mole completely and causing undue damage to the uterus by continuing the curettage. With due consideration to both sides of this question, it is safe to say that with mole the uterus is seldom as completely emptied at curettage, and probably should not be, as in the case of incomplete abortion.

The probability that portions of the mole are left behind to continue growth or to undergo malignant change necessitates careful observation of the patient for at least 2 years. The most important points to watch are the patient's menstrual behavior, the size of the uterus and the Aschheim-Zondek test. The test should have been repeated when the patient reported to the postnatal clinic after the uterus was emptied. Her family physician had no Aschheim-Zondek test done from the time of her discharge from the first hospital to the time of her admission to the second, at which time malignant changes were of long standing and metastases were widespread throughout the body.

After a mole, the patient is entitled to receive protection against the risk of malignancy. This risk is about 7 per cent. The best protection we can give at the present time includes repeated examination and repeated Aschheim-Zondek tests over a period of 2 years and immediate hysterectomy followed by radiation if signs of malignancy are found.

MISCELLANY

APPOINTMENTS AT HARVARD MEDICAL SCHOOL

Nine appointments to the teaching staff of the Harvard Medical School, effective now and continuing until next September 1, were announced February 24, as follows:

Anders J. W. Hagstromer, M. K. Karolinska Institutet, Stockholm, '32, research fellow in surgery.

Edward H. Holling, M.R.C.P. Royal College of Physicians, London, '36, research fellow in surgery.

John C. Snyder, M.D. Harvard '35, research fellow in surgery.

John E. Dunphy, M.D. Harvard '33, assistant in surgery.

Jesse L. Conel, Ph.D. Illinois '16, research fellow in pathology.

Sylvester McGinn, M.D. Harvard '29, instructor in medicine.

Roy B. Cohn, M.D. Stanford '33, assistant in surgery.

Kikuo Kurauchi, Ph.D. Keio University '33, research fellow in bacteriology and immunology.

CONNECTICUT NEWS

Report of Temporary Commission to Study Financial Responsibility Act

The Commission appointed by Governor Cross in December, 1936, has made its recommendations. It presents essentially the plan that was recommended by the Legislative Committee of the State Medical Society, and includes the setting up of a State fund. The following paragraphs are quoted from the report:

"We recommend that a State fund be established, and that from this fund the medical, surgical, hospital and nursing expenses be paid for every resident of Connecticut injured by an automobile upon a public highway in the State of Connecticut, in a sum not exceeding three hundred dollars. We suggest the maximum of three hundred dollars, but we know that in many cases the damage will exceed three hundred dollars. In order that the fund may insure the immediate and continued treatment of injured persons, our suggestion is that it be administered expeditiously and without delay and, particularly, without any reference to whether there is fault or ultimate recovery on the part of the injured person. As we have previously indicated, if it were necessary to wait for a determination as to fault or liability, then the injured person would be deprived of the essential, immediate and continuous treatment.

"Such bill ought to contain a provision that the injuring motorist be required to compensate the fund for the amount paid by it on behalf of the injured person, and that, on failure of the injuring motorist to compensate the fund, the license or registration—or both—of the motorist and his vehicle should be suspended, and, in the event of his continued failure for a definite period of time to compensate the fund, then that license or registration certificate—or both—should be taken away from the injuring motorist.

"The amount of money so paid from the State fund should be returned to the State fund by the injuring motorist before any payment is made to the person for whose benefit the expenditure was made from the State fund.

"We do not submit a draft of such bill, as we realize there are many elements upon which reasonable men may not agree. We are entirely conscious of the intent of the legislature in appointing this committee and, if a legislative committee deems it advisable to confer with us about such a bill, we will be very glad to render such services as lie within our power."

Hartford Medical Society Surgical Section

At the annual meeting held in Hartford on January 25, Dr. Benedict B. Landry was elected president; Dr. Welles A. Standish, secretary, and Dr. Joseph Heyman, treasurer.

Hartford Tuberculosis and Public Health Society

Dr. Herbert R. Edwards, director of the Bureau of Tuberculosis of the New York Health Department, spoke on "Coordinated Tuberculosis Control" at the annual meeting, January 27. The following officers were elected for 1937: Dr. Edward H. Truex, president; Dr. David Gaberman and Mrs. Otto G. Wiedman, vice-presidents; Miss Mary A. Readett, secretary; Louis A. Allyn, treasurer.

New Hospital for Southington

Edwin S. Todd, president of the Hospital Corporation of Southington, announced recently that preliminary plans for the Bradley Memorial Hospital, which is to be erected at Meriden Avenue and Oakland Road, have been agreed upon by the Hartford National Bank and Trust Company, the Hospital Corporation of Southington and the State Department of Health.

The preliminary plans were drawn by Alexander S. Lindsay of Southington, consultant architect for the Hospital Corporation. It is understood that Lester B. Scheide, Inc., of Hartford, will soon furnish detailed specifications and drawings of the building. It is estimated that the building when fully equipped will cost about $100,000. The proposed building is to be of fireproof construction and will have three floors—basement, first floor and second floor. The area of each floor will be about 4,200 square feet. The west side of the L-shaped structure will be about 80 feet in length and will face Meriden Avenue, and the south side facing Oakland Road will be about 84 feet in length. The basement will have garage space for an ambulance and three automobiles, an accident room, special clinic rooms for social diseases, laundry, boiler and other rooms. The first floor space will be devoted to public health work of various kinds, preventive and otherwise. An assembly room or auditorium will be located on the first floor. The second or top floor will be given over to emergency hospital facilities, including a complete operating unit, two small wards, two private rooms for patients, nurses quarters, and so forth. Living quarters, kitchen and dining rooms for janitor, cook, and so forth, are included in the building.

The Hospital Corporation has been at work for the past few months grading and preparing the grounds for the new building. More than 12,000 cubic yards of fill have been removed, and new sidewalks, gateway posts and a retaining wall of Stony Creek granite have been built. The old dwelling house on the site will be torn down and removed.

Mr. Todd expects that work will be started on the new building in the early part of the summer. The Hospital Corporation Fund exceeds $425,000 at present.

Dr. Long Addresses Hartford Medical Society

Dr. Perrin H. Long, of Baltimore, addressed the Hartford Medical Society, February 1, on "pron-

tylin". Dr. Long is an associate in medicine at the Johns Hopkins University School of Medicine. He has been engaged in research work on streptococci and spent last summer in England where he made a special study of prontylin. A capacity audience was present.

Hartford Hospital Receives Bequest

The will of Dr. William D. Morgan, of Hartford, directs a bequest of $5,000 to the Hartford Hospital in memory of his parents.

Group Hospital Insurance Advocated

Dr. Wilmar M. Allen, director of the Hartford Hospital, in his annual report suggests voluntary group hospital insurance to meet the problem of hospital costs to people of moderate means. Dr. Allen believes that this arrangement would help to solve the problem of the abuse of medical charity by a relatively small but significant number of people. Group hospital insurance is now operating effectively in many communities. It has been discussed by the Hartford Medical Society and is now being studied by the Hartford County Council on Hospitals.

RUSSELL—George W. Russell, M.D., of Waterbury, died February 4 at St. Mary's Hospital, after a short illness. He was in his sixty-fifth year.

Dr. Russell had been in active practice for almost forty years. Many of these years he was chief of the staff of St. Mary's Hospital. He served a number of terms as president of the Waterbury Medical Association and was a member of the county and state societies.

He leaves a widow, Ellen (O'Mara) Russell, seven daughters and a son.

CORRESPONDENCE

REREGISTRATION

February 24, 1937.

Editor, *New England Journal of Medicine*,

I feel that the bill for annual registration of physicians has an object entirely apart from seeking out unlicensed physicians. This bill requires that the physician shall file certain information, which the Board may publish and circulate in lists of registered physicians. The object of this is to guide the prospective patient to members of the Massachusetts Medical Society, to graduates of schools approved by the American Medical Association and, in the case of the specialties, to members of certain societies. I do not believe the Board should be given the power that might be used to discriminate against duly registered physicians who are honestly practicing their profession. The Board already has the power to revoke a license for just cause, and I think this is enough. About one-third of the registered physicians would be discriminated against and would incidentally be taxed to carry on this discrimination. When the Board asks to publish more than a simple list of registered physicians, it can only be for the purpose of discrimination. I do not believe it should be a part of the function of the Board directly or indirectly to discriminate between duly licensed physicians, regardless of school of graduation, society membership or system of practice.

The certificates and diplomas in the physician's office give the information that the Board wishes to publish at the physician's expense. While the school diploma is in Latin, the certificate of registration gives the name of the medical school in English as well as the date of graduation and registration. The certificate of the Massachusetts Medical Society is in English. The same is true of the certificate of the American College of Surgeons. Most certificates of hospital internship are in English.

Yours very truly,

Milman Pease, M.D.

Brookfield, Mass.

RECENT DEATHS

MAGUIRE—Eugene Leo Maguire, M.D., of 390 Medford Street, Somerville, died at his home, February 26, 1937, in his fifty-ninth year.

A native of Somerville, Dr. Maguire received his early education in the public schools of that city, attended Holy Cross College for two years and, later, Dartmouth College, where he received his A.B. degree in 1901. He was graduated from the Harvard Medical School in 1905 and served his internship at the Carney Hospital. Later he was house physician at the Boston Lying-in Hospital. He had maintained offices at 390 Medford Street since 1907. During recent years he had been Senior Surgeon at the Somerville Hospital.

He was a member of the Massachusetts Medical Society, the American Medical Association, the New England Obstetrical and Gynecological Society, the Somerville Medical Association and Sigma Chi fraternity.

His widow, Mrs. Mary Maguire; a daughter, Kathleen, and two sons, Eugene, Jr., and Walter, survive him.

O'BRIEN—Daniel Paul O'Brien, M.D., of 330 Union Street, New Bedford, died at New London, February 27, 1937, after a short illness. He was in his fifty-ninth year.

A native of Chelsea, Dr. O'Brien was graduated from the Harvard Medical School in 1904. He was a member of the Massachusetts Medical Society, the American Medical Association, the American College of Surgeons and the New England Surgical Society.

His widow and two children survive him.

OBITUARY

RESOLUTIONS BY THE STAFF OF THE QUINCY HOSPITAL UPON THE DEATH OF LAWRENCE PEARS CRAWFORD

Whereas, Dr. Lawrence Pears Crawford, our associate in the practice of medicine in this community for nineteen years, has been removed by death, and

Whereas, the Staff of the Quincy City Hospital feel keenly the loss of one who was devoted to his patients through his methods, conscientious in all his professional relations, and a loyal friend, and

Whereas, we desire to extend our sympathy to his widow, Mrs. R. Dorothea Crawford, and children:

Therefore, be it resolved that this resolution be entered upon our records and copies be sent to Dr. Crawford's family and to *The New England Journal of Medicine.*

GEORGE D. DALTON, M.D., *Staff Secretary.*

REPORTS OF MEETINGS

THE WACHUSETT MEDICAL IMPROVEMENT SOCIETY

A special meeting of the Wachusett Medical Improvement Society was held on December 22, 1936, at the Holden District Hospital.

The meeting was called to order at 8:15 p. m. by Dr. Crane, president of the society.

DR. CRANE: In the two previous lectures that Mr. Proctor has given us, he has talked of "Parliamentary Procedures" at the first meeting, and "Motions," at the second meeting. Tonight he is going to give us a lecture on "Procedures at Meetings."

MR. PROCTOR: *Mr. President*—This is the third and final lecture of the course in lecture form. I thought that we would try to conduct the next meeting according to parliamentary practice. I will select one of the town warrants and see what can be done in the way of a few of the articles there.

PROCEDURE AT MEETINGS

PRIVILEGED QUESTIONS

Privileged questions are matters of superior importance that take precedence over subjects under consideration by the meeting, and that must be disposed of before the matters then under consideration are further discussed. They have a different order among themselves and may be divided into three classes: first, motions to adjourn; secondly, motions or questions relating to the rights and privileges of the meeting or of its members individually; and, thirdly, motions for the orders of the day.

Motions to adjourn take precedence over all other questions, but the motion to adjourn must be made without stating a particular day or hour. A motion to adjourn made pending the discussion of another matter is not subject to an amendment and is not debatable, but, if qualified to adjourn to a certain time or day, it is debatable. The effect of an unqualified motion to adjourn is an adjournment to the next regular sitting or to a day that has been previously decided upon.

It has been officially decided that the chairman is not bound to put such a motion, but, of course, the common practice is otherwise. If a motion to adjourn is made pending discussion of another matter, the other matter is not in order at the next meeting unless specially called for.

ORDERS OF THE DAY

Orders of the day are subjects that are assigned for discussion on a particular day and sometimes at a particular hour, and, if no hour is assigned, they are taken up in the precedence in which they appear on the calendar or other memoranda of the meeting.

If business is assigned for a particular hour, when that hour arrives, a motion for the orders of the day will supersede motions then before the meeting, and the business assigned is taken up, leaving the previous matter undisposed of. This, of course, does not apply to a motion to adjourn, whether the motion is to adjourn to a certain day or otherwise.

A motion to proceed to the orders of the day means to proceed generally and not to proceed to take up any particular matter, and if the motion is carried, it is the duty of the chair to read all matters placed on the calendar before the first is taken up.

A matter that is interrupted by a vote to proceed to the orders of the day is not *ipso facto* before the meeting when the orders of the day have been completed, and a motion must be made to take it up.

If the motion to proceed to the orders of the day is voted down, the orders of the day have no standing if not proceeded to on the day on which they are assigned, and must be moved separately as they have no particular preference.

READING PAPERS

When papers have been read before a meeting, every member has a right to have them read once at the table before he can be compelled to vote on them. Hence, if a member calls for their reading, the papers are read by the clerk under the direction of the presiding officer as a matter of course. If papers are not laid before the meeting but are only incidentally involved, a member has no right to read them himself or to have them read, as this would cause undue delay and interruption. Hence, if a member desires to have papers, books or documents read or desires to read them, or even if he wishes to read his own speech that he has prepared beforehand and committed to writing, he must obtain leave of the meeting by motion and vote for that purpose. It is the usual practice of the chairman, however, to permit the reading of papers unless an objection is made, or unless it is apparent to him that their reading is not worth the time involved or is intended for delay. A motion to have a paper read, of course,

is an incidental motion and should be disposed of before the main motion is put.

SUSPENSION OF RULES

When a motion or proceeding is rendered impossible or impracticable by reason of a special rule prohibiting it, it is permissible to suspend or to dispense with a rule for the purpose of admitting the proceeding or the motion that is desired. The rule can be suspended only by motion and vote, hence, a motion to suspend the rule takes precedence over the question pending before the meeting. In the absence of by-law or other prohibition, the rule can be suspended only by unanimous vote, but it is competent for the meeting or body to pass a by-law that qualifies a smaller number of votes to suspend the rule.

Matters laid on the table may be taken up again at a subsequent time, and no motion is necessary for this purpose since the call of any one member is sufficient.

AMENDMENTS OF SUBSIDIARY MOTIONS

As a general rule, amendments of subsidiary motions are not subject to amendment, but there are certain exceptions, among which are the following: motions to commit or to recommit to committees may be amended by the substitution of one committee for another, by enlarging or diminishing the number of members of a special committee or by instructions to the committee. Unlike most motions of a subsidiary nature, a motion to commit is debatable and subject to amendment. It takes precedence over a motion to amend, and, once moved, the previous question cannot be applied to it, nor can a motion to postpone be applied to it. If decided affirmatively, it is removed from the meeting, hence, there is no ground for the previous question, and, if decided negatively, the main motion is then open to subsidiary motions as if the motion to commit had not been made. A motion to amend may be superseded by a motion to commit or to recommit.

The following rules apply to the following motions and other matters arising during debate:

Of Main Motions

1. A speaker may not be interrupted.
2. Requires a second.
3. Debatable.
4. Majority vote required.
5. Motion may be renewed at next session.
6. All motions may apply.

Of Amendments

1. A speaker may not be interrupted.
2. Requires a second.
3. Debatable.
4. Majority vote required.
5. Motion may not be renewed.
6. Motions that may apply are: Amend — Limit debate—Previous question—Reconsider.

Of Point of Order

1. A speaker may be interrupted.
2. No second required.
3. Not debatable.
4. If not decided by chairman, a majority vote is required.
5. Motion may not be renewed.
6. No motions may apply.

Of Request for Information

1. A speaker may be interrupted.
2. No second required.
3. Not debatable.
4. No vote required.
5. May not be renewed.
6. No motions apply.

Of Previous Question

1. A speaker may not be interrupted.
2. Requires a second.
3. Not debatable.
4. Two-thirds vote required.
5. May be renewed after debate.
6. Motion to reconsider may apply.

Of to Limit and Extend Limits of Debate

1. A speaker may not be interrupted.
2. Requires a second.
3. Debate limited.
4. Two-thirds vote required.
5. May be renewed after debate.
6. Motions that may apply are: Amend—Reconsider.

Of to Refer to a Committee

1. A speaker may not be interrupted.
2. Requires a second.
3. Debate limited.
4. Majority vote required.
5. May be renewed after debate.
6. Motions that may apply are: Amend — Limit debate—Previous question—Reconsider.

Of Question of Privilege

1. A speaker may be interrupted.
2. No second required.
3. Not debatable.
4. No vote—chairman decides.
5. May be renewed after debate.
6. All motions may apply.

Of Resolutions

1. A speaker may not be interrupted.
2. Requires a second.
3. Motion is debatable.
4. Majority vote required.
5. May be renewed at next session.
6. All motions may apply.

Of Appeal from Decision of Chair

1. A speaker may be interrupted.
2. Requires a second.
3. Debate limited.
4. Majority vote required.
5. May not be renewed.
6. All motions apply except amend.

Of to Lay on Table

1. A speaker may not be interrupted.
2. Requires a second.
3. Not debatable.
4. Majority vote required.
5. May be renewed after debate.
6. No motions apply.

Of to Take from the Table

1. A speaker may not be interrupted.
2. Requires a second.
3. Not debatable.
4. Majority vote required.
5. May be renewed after business has intervened.
6. No motions apply.

Of Postpone Indefinitely

1. A speaker may not be interrupted.
2. Requires a second.
3. Debatable.
4. Majority vote required.
5. May not be renewed.
6. Motions that apply are: Limit debate—Previous question—Reconsider.

Of Postpone Definitely

1. A speaker may not be interrupted.
2. Requires a second.
3. Debate limited.
4. Majority vote required.
5. May be renewed after debate.
6. Motions that may apply are: Amend — Limit debate—Previous question—Reconsider.

Of Create Orders of the Day

1. A speaker may not be interrupted.
2. Requires a second.
3. Debatable.
4. General—majority special two-thirds vote required.
5. May be renewed after debate.
6. All motions apply.

Of Call for Orders of the Day

1. A speaker may be interrupted.
2. No second required.
3. Not debatable.
4. No vote required.
5. May be renewed after business pending is disposed of.
6. No motions may apply.

Of Object to Consideration

1. A speaker may be interrupted.
2. No second required.
3. Not debatable.
4. Two-thirds negative vote required.
5. May not be renewed.
6. Motion to reconsider may apply.

Of Suspend Rules

1. A speaker may not be interrupted.
2. Requires a second.
3. Not debatable.
4. Two-thirds vote required.
5. May not be renewed except by unanimous consent.
6. No motions apply.

Of Without a Motion

1. A speaker may not be interrupted.
2. No second required.
3. Not debatable.
4. Majority vote required.
5. May be renewed after debate.
6. Motion to reconsider may apply.

Of to Adjourn

1. A speaker may not be interrupted.
2. Requires a second.
3. Not debatable.
4. Majority vote required.
5. May be renewed after debate.
6. No motions apply.

Of Fix Time to Which to Adjourn

1. A speaker may not be interrupted.
2. Requires a second.
3. Limited debate.
4. Majority vote required.
5. May not be renewed for some time.
6. Motions that may apply are: Amend—Reconsider.

Of Take a Recess

1. A speaker may not be interrupted.
2. Requires a second.
3. Debate limited.
4. Majority vote required.
5. May be renewed after business has been disposed of.
6. Motion to amend may apply.

Of Reconsider

1. A speaker may be interrupted.
2. Requires a second.
3. Debatable.
4. Majority vote required.
5. May not be renewed.
6. Motions that may apply are: Limit debate—Previous question — Postpone definitely—Lay on table.

Of Reconsider and Have Entered on the Minutes

1. A speaker may be interrupted.
2. Requires a second.
3. Not debatable.
4. No vote required until called up.
5. May not be renewed.
6. No motions may apply.

Of Rescind

1. A speaker may not be interrupted.
2. Requires a second.
3. Debatable.
4. Two-thirds vote required.
5. May not be renewed.
6. All motions may apply.

Of Expunge

1. A speaker may not be interrupted.
2. Requires a second.
3. Debatable.
4. Two-thirds vote required.
5. May not be renewed.
6. All motions may apply.

ORDER OF BUSINESS

When there are several matters pending and none of them have actually come before the meeting, the presiding officer is not bound to any order as to which matter should be taken first, but may proceed at his own discretion. Where the meeting is apt to sit for a considerable time, however, it is useful to establish and settle the order of business, which may be done by general rule or by special orders relating to particular subjects.

PROCEDURE ON COMMITTEE REPORTS

The first business in order is to take up the amendments that have been submitted by the committee and so on until the whole are either reported or rejected, before any other amendment is admitted, with the exception of an amendment to an amendment.

After these have been disposed of, amendments to the main subject are in order, and when these have been disposed of, the question of agreeing to or adopting the paper, as the resolution, order, and so forth, of the meeting is put. The report should be accepted, and it is better form to agree to its substance by name rather than to leave the matter hanging on a mere acceptance of a report. When a committee reports in amendment form, the new draft is considered as an amendment and, if necessary, is first amended, and then put to the question as an amendment reported by the committee, or the new draft may be accepted as a substitute for the original paper and then treated as a motion before the meeting.

PENDENCY OF SEVERAL MATTERS

When several matters are pending at the same time, a principal motion and a subsidiary one related to it should not be proposed and stated together, but the subsidiary one must be disposed of before the main one is put. When a member makes a principal motion and then follows it immediately with a subsidiary motion as, for instance, concerns a previous question, no attention should be taken of his subsidiary motion as it is out of order.

PUTTING THE QUESTION

A motion is the method of putting a matter before a meeting by a member. A question is the stating of the motion by the chair for decision by the meeting, and the decision of the meeting becomes the order, resolution or vote of the meeting. The question and the vote will now be discussed.

After debate the chair may ask the meeting if it is ready for the question, and if no person rises, the question is then stated, and the vote of the meeting is taken on it in several methods:

(a) By silent assent.
(b) By voice.
(c) By holding up hands.
(d) By division.
(e) By roll call.
(f) By ballot.
(g) By yeas and nays.

(a) By silent assent. This method is used where routine matters are presented or matters of trifling importance, such as receiving petitions, reports, and so forth. The chair declares that no objection is made and declares a vote to receive the matter, although none has been in fact, taken. If an objection is made, however, it is the duty of the chair to put the matter to a formal vote.

Where any of the other methods of voting are employed it is the duty of the chair to put the question in the affirmative first and then to put it in the negative, employing the words, "As many as are in favor" or "Those that are of the opinion that," and so forth and indicating the method of the taking of the vote.

The most common method of voting, where only a majority vote is required, is by voice (b), and the chair decides by the volume of sound. The use of the voice vote is not recommended where more than a majority vote is necessary, because of the difficulty in distinguishing whether the required fraction has been ascertained. If the chair is in doubt, he may put the question a second time, or any person may rise and doubt the vote, by using those words or, by saying that he believes the "ayes" have it when the chair has declared otherwise, and so forth. If new matter is taken up before the vote is doubted, either by the chair itself or by a member of the meeting, it is then too late to question the vote, and it is also too late to question the vote should a member of the meeting who has been previously absent come in.

Where the vote has been by voice, the chair may in its option or on call of a member ask for voting by a show of hands or by division or otherwise, but a member has no right to call for another method of voting than by a voice vote or a vote by division unless the articles under which the meeting is being conducted provide otherwise.

A division may be had in any of the following methods: first, by asking the members to arrange themselves on different sides of the assembly room where they are counted, either by the chairman, or by tellers appointed by him, or by monitors appointed for that and other purposes; or the chair may, and more commonly does, ask the members to rise in their seats, first, those on the affirmative, and then those on the negative, when they are counted in the same manner.

When counted by tellers or monitors, the tellers must first agree among themselves as to the count, and then report it to the chair, or monitors may be appointed to count a part of the meeting, in which

case, those to whom a part only has been assigned must agree among themselves as to that part.

When the report of the tellers or monitors has been heard, it is then the duty of the chair to declare the final vote.

The chair has a right to vote on all questions, but it is a right to be used sparingly, and it is considered as an attempt to influence the members unduly if he votes on matters where his vote, if taken by itself, would not change the result. When a matter is about to be carried by one affirmative vote and the chair desires to vote in the negative, he may vote or refrain from voting as good sense dictates. Where a vote is a tie it is his duty to vote in any event. Under the rules of the United States House of Representatives, the speaker is required to cast his vote if it would be the deciding vote, and this has been held, according to the House, to be:

(a) When necessary to break a tie.
(b) To create a tie.
(c) To complete a two-thirds vote.
(d) To make a quorum.

In the United States Senate the Vice-President presides and has no right to vote except in the foregoing instances, and the same is true of the Senates of many of the states, although not in Massachusetts.

All other members that are in the room at the time should vote, and no one that is not in it has a right to vote. Should all members keep silent when both the affirmative and negative are put, the vote is taken as a unanimous affirmative vote.

TAKING THE QUESTION BY AYES AND NAYS

Under this method the question is put only once, and each member is called by name. The member rises in his seat and says either "Yes" or "No," and the clerk records the answers that are made. It is his duty then to read the names of those voting in the affirmative and, after he has read the names of all those voting in the affirmative, to read the names of all those voting in the negative in order to avoid error, and then to tabulate the vote and to notify the chairman of the result. The chairman then announces it to the meeting. It is sometimes the custom, instead of reading the whole list through a second time, for the clerk to repeat the name of the member after he has voted with the way he votes.

Where a question is put, first, in the affirmative form, and secondly, in the negative form, it is not completed until the negative vote has been taken as well as the affirmative vote. Hence, it is in order for a member to rise in his seat and to make motions concerning the question or to debate it, even after the affirmative vote has been taken. This is the case whether he was in the room at the time the question was first put or not. If he does so, the question must be put over again, on the affirmative as well as on the negative side, for the reason that the minds of the members may have been changed by what has been said or members may have entered or left during the discussion. When a question is taken by yeas and nays, so that the voting on each side proceeds at the same time, the question cannot be opened after the vote has commenced, and the same is true when the voting is by ballot, because this is another method in which the voting on both sides begins at the same time.

If a question arises on a point of order during the division as, for instance, the right of a person to vote, the presiding officer must decide it pre-emptorily, his decision being open to review after the division is over. Before making his decision, however, the presiding officer has a right to call upon any member of the meeting for advice, but it is the duty of such a member to sit down before answering the presiding officer and to remain seated to avoid even the appearance of debate or partisanship.

If, upon division, it appears that there is not a quorum present, no vote is taken, and the matter is taken up again, the question before the meeting being the same as it was at the time that the question was first propounded.

RECONSIDERATION

A matter once voted by a meeting cannot be voted upon again, either directly or indirectly, except by reconsidering the whole matter. A motion for reconsideration recognizes and upholds the rule that a matter, once passed, is the will of the meeting, yet permits it to relieve itself of the embarrassment and inconvenience that results from a strict enforcement of a rule in a particular case.

After a motion for reconsideration is made and carried, the matter stands before the meeting in the same state as if the vote had never been passed. If a vote to reconsider is made and lost, the matter cannot subsequently be reconsidered, and the motion to reconsider a vote taken on the question of reconsideration is not in order.

Where the main motion has been amended and finally put to a vote and the motion, as amended, has been carried, a reconsideration cannot be had on the votes on the amendment without first reconsidering the question in its amended form. The practice in such an instance is, first, to reconsider the last vote, that is, the vote passing the main question in its amended form, and, if this is carried, then to move to reconsider the question on the amendment. This applies equally to a case in which the original vote on the amendment was in the negative.

COMMITTEES AND COMMITTEE REPORTS

Committees are appointed to consider matters that the meeting, itself, has not time to consider, to take preliminary measures, to prepare reports and to do other things that in their nature cannot be done during the course of deliberative assemblies. Committees that have been appointed for specific matters are called select, and committees that have been appointed beforehand for all matters of the same nature are called standing committees. Their powers and functions depend upon the authority

and particular instructions given them by the meeting at the time of the appointment, but these can be added to, limited or otherwise changed by instructions given them from time to time by the body of which they form a part.

In a vote to appoint a select committee, the first step is to determine the number of members, and if several different numbers are proposed, each number is put to a vote separately, beginning with the largest and going through to the smallest, until the meeting finds a number upon which it can agree. When the number is determined, the members may be appointed by the presiding officer, by ballot or by nomination and vote of the meeting. The motion providing for the committee usually defines the method of appointment, but if no vote is taken, and no rule governs, the members of the committee should be nominated from the floor. It is customary for by-laws or other regulations to provide for their appointment by the chair. If such a rule exists, and if the meeting desires the committee to be appointed otherwise, it may do so by vote. In case of an appointment by ballot, the vote is taken as in the election of officers. Where the appointment is from the floor, the presiding officer calls upon the meeting to nominate, and he places each name before the meeting in the order in which he hears the nominations received. It is also proper to revive a committee that has already been discharged, and to refer a second matter to a committee that has already been appointed in a previous matter. It is a general rule for the guidance of the chairman and of the meeting itself, not to appoint to a committee members that have opposed the matter under discussion, as it is the duty of the committees to facilitate the matter rather than to oppose it, but in this the meeting or the chairman is the final judge.

After a committee has been appointed, the secretary of the meeting should make out a list of the members and a certified copy of the authority or instructions under which they are to act, and give the papers to the member that is first named on the committee, if convenient, or to any other member, if it is not convenient to give it to the person first named.

The first named member of the committee acts as chairman or presiding officer for the purposes of organization. After organization, his continuance as chairman is a matter of courtesy, and every committee has a right to elect its own chairman, who acts as its presiding officer and usually makes a report of its proceedings to the meeting.

A committee is essentially a miniature assembly in its proceedings and can act only when regularly assembled together as a committee. Votes should be taken in committee. A quorum is necessary and is a majority unless a smaller number has been fixed by the main meeting itself. Unless its powers are limited, it has full power over whatever has been committed to it, except the right to change the title or the subject. Unless otherwise ordered, it may meet when it pleases and adjourn to such times as it sees fit, but if it is ordered to meet at a particular time and fails to do so, the committee is closed and must go again to the body that appointed it for further instructions. It has no power to punish its members for disorderly conduct, but may report such matters to the meeting for disciplinary measures.

When the committee has either originated papers or has had papers referred to it, such papers should be read entirely through by the clerk of the committee, or by the chairman, if there is no clerk, and then should be read again by paragraphs by the chairman, who should pause at the end of each paragraph and put questions for amending, if any are proposed, but the same strictness that should be followed in a meeting is not necessary in a committee meeting.

The paper should be amended paragraph by paragraph, but the paragraphs themselves are not adopted in their amended form seriatim, as this is reserved for vote after the whole paper has been considered. The committee has no final question on the adoption of the whole paper where a paper has been referred to it because this is a matter for the meeting to decide. Even if they are opposed to it in its entirety and feel that it cannot be made good by amendment, they have no authority to reject it but must report it without amendment, specifying their objections to the matter. Of course, if subject matter, instead of a paper, is referred to a committee, the committee has authority to say that it thinks that no action should be taken on the subject and otherwise to disapprove of the subject.

In the case of a paper that originates with the members of a committee, they may erase or interline it as they please, although when the report is finally agreed to, it should be in a clear draft, without erasure or interlineation. In the case of a paper that is referred to a committee the members are not at liberty to change it at all, but may in a separate paper set down the amendments that they have agreed to report, stating the words that are to be inserted or omitted in places where the amendments are to be made by reference to the paragraph or section, line and word. If amendments are numerous, the committee may report them together in the form of a substitute draft.

After the business is completed, a motion that the committee rise and that the chairman or some other member make the committee report to the meeting is in order. The committee should not sit when the meeting itself is in session, and if advised of the session it is the duty of the committee to rise, attend the meeting and return to its work after the meeting is over.

COMMITTEE REPORTS

When the committee is ready to report, the chairman or other reporting member stands in his place and informs the meeting that the committee has a report to make when the meeting shall be ready to receive it, and it may be moved that it now be received. This motion may be made by the

reporting member or by any member of the meeting, whether a member of the committee or not, and if the vote is passed, the meeting then receives the report. The meeting may vote, however, not to receive it then, to receive it at some future time or not to receive it at all.

When the time arrives for the report to be received, the chairman reads it in his place and delivers it in its written form, with the papers connected therewith, to the clerk at the table where it is again read. It lies on the table until it suits the meeting to take it up for consideration. In practice, the formality of motion and vote to receive a report is usually dispensed with, but if an objection is made, such a vote is necessary, and the chairman should not receive the report over an objection except by vote of the meeting. After the report is received the committee is dissolved and has no further power to act unless again requested to do so by another vote of the meeting. If the report is not received, the committee is not discharged but may be ordered to sit again, and the meeting may assign a time and place for its sitting. After receiving a report, the meeting may recommit it to the same committee or to a different committee, and if it is recommitted before it has been agreed to by the meeting, what passed before is of no force. The entire question is again before the committee as if nothing had passed in reference to it.

A report may be made in three forms: first, it may contain merely a statement of facts, reasoning or opinion in relation to the subject of the report without specific conclusion, or, secondly, it may contain a statement of facts, reasoning or opinion concluding with a resolution or series of resolutions or some other specific proposition, or, thirdly, it may consist merely of such resolutions or propositions without any introduction.

Once a report is before the meeting, the following procedure is in order:

(a) If the report relates only to an opinion or statement of fact, and no motion is made by the reporting member for its disposal, the report is merely accepted, which means that the meeting assumes the responsibility for the contents of the report. A report that is related to finance should be submitted to an auditing committee, as the meeting cannot guarantee the accuracy of figures, and if there is no auditing committee, one should be appointed by the chair.

(b) Recommendations in a report which are not in the form of motions should appear at the end of the report and another motion should be made to adopt the recommendations, even though they have been previously given separately.

(c) If a report contains resolutions, the reporting member should move that the resolutions be adopted.

(d) The recommendation of the committee determines the form of the question to be stated by the chairman, first, if the committee can come to no agreement and makes no recommendation or when it does recommend its adoption, the question should be stated on the amendment, if one should be pending, and then on the resolution. Motions should not be repeated as they were pending when the motion was referred to the committee. Secondly, when the recommendation is against the adoption of the resolution, the form in which the question should be stated is, "The question is on the adoption of the resolution, the recommendation of the committee to the contrary notwithstanding" and the same is true on the amendment where the committee has reported adversely. Thirdly, when a resolution or a paper with amendments is reported by the committee, the reporting member should read the amendments only, sufficiently in detail to render them clear, and then their adoption should be moved, the chair then stating the question on adoption and directing the first amendment to be read, which is then open for debate and amendment. After a vote is taken on this amendment, the others are ready in turn until all amendments recommended by the committee have been disposed of, admitting amendments by the meeting only to those made by the committee. After the committee's amendments have been disposed of, other amendments may be proposed by the meeting and after being voted upon, the question is put by the chairman as to agreeing to or adopting the paper as amended, unless it has already been adopted.

Of course, the foregoing rules apply only in cases where parliamentary practice is followed strictly, and the report may be adopted by general consent or suspension of the rules, notwithstanding what has previously been said. It is also proper when the amendments are formal or call for no debate or amendment, for the chair to put a single question in all the committee's amendments, unless a member requests a separate vote in a specific case. The question then is, "All those in favor of adopting the amendments recommended by the committee (except such amendments on which a separate vote has been called for) will say, 'Aye'." Having disposed by the omnibus vote of the amendments that are not questioned, the remaining amendments are taken up seriatim.

If a resolution has been committed, and the committee desires to report a substitute, the chair states the question on the substitute, but if there were amendments to the resolution pending at the time of the commitment, then those amendments that were so pending are first disposed of before the question on substituting the committee's report for the original resolution is taken up.

After these have been disposed of, the motion is made to substitute the committee's report for the original resolution, and at that time the substitute resolution is open for amendment by the meeting. After the amendments to the substitute resolution have been disposed of, the vote is then put on substituting the resolutions as amended. If that motion is carried, the original resolution, of course, disappears, but if the motion is lost the original reso-

lution is again before the meeting for further amendment and discussion, if desired.

The report of a nominating committee requires no vote to adopt it, and the names are before the meeting as though the nominations had been made from the floor of the meeting itself. When a membership committee reports, no vote is required to accept it, and the chair proceeds to state the question as to the admission to membership of candidates that have been recommended.

Incomplete and complete reports of committees are treated alike, but when the incomplete report refers only to progress, no action is called for unless the report specifically recommends it.

In the absence of a motion to accept or to debate the report of a committee, the chair may state the question, if he considers it best to have the question voted upon before any motion is made, and if no objection is made to this procedure the vote is taken forthwith.

Like other actions of a meeting, reports of committees are subject to subsidiary motions such as to lay on the table or the previous question. The meeting has no power to change the report itself, but may vote to amend or otherwise change votes relating to the report. If the report is published, it must be printed verbatim as it was submitted. Words that have been inserted by the action of the meeting may be printed in italics, and words that have been stricken out by the action of the meeting, enclosed in brackets.

DISCUSSION

MR. PROCTOR: I think that is all that we have for tonight. I will be glad to answer any questions that you gentlemen may have, either about this lecture or any of the other lectures, or concerning any matter that we have not discussed.

DR. CRANE: Are there any questions?

MEMBER: You stated that a committee had the privilege of choosing its own chairman. May the presiding officer appoint his own chairman?

MR. PROCTOR: The rule is that he may, but his powers may be superseded by the committee, which may appoint a new chairman.

MEMBER: Will you kindly tell us what executive session means?

MR. PROCTOR: It is an occasion where the executive committee meets and sits around the table and talks informally, and does not proceed by any parliamentary practice at all, and they finally end up by getting some sort of a vote. It is supposed to be secret session, but it is permissible for them, if they so desire, to ask some person, who may not even be a member, to come before them and to assist in the meeting.

MEMBER: When a body is in executive session, do the proceedings appear in the secretary's report?

MR. PROCTOR: No, they are not supposed to be published in any way.

MEMBER: You were saying that when a committee was appointed, the members of the committee were supposed to be in favor of the measure to be considered, and that the chairman was judge of that matter.

MR. PROCTOR: If the chairman appointed the committee, it is up to him to decide. In the small duties that are provided to me as Moderator in the town, I like to select people that have minds on both sides of the question.

MEMBER: That then is the duty of the chairman, whether or not he is in favor of it.

MR. PROCTOR: It is the act of a wise chairman not to express his opinion in such matters.

MEMBER: Tell us something about the changing of the constitution and by-laws.

MR. PROCTOR: Changing the constitution is usually provided for in the by-laws themselves. The amendment should be submitted in writing and should be laid on the table for a certain number of meetings and a written notice sent to all the members. I have never yet seen a constitution or by-laws that did not take care of this provision. Without there being such a provision in the by-laws, you would have to amend them by majority vote, but you could amend the constitution only by a unanimous vote.

MEMBER: Suppose the proposed change lay over for a future meeting, can those proposed changes be altered and then made at that meeting? Is it legal?

MR. PROCTOR: It is not legal if you change the intention, but if you change the words or make mere clerical changes, it is all right. If the substance itself is changed it should be proposed again, and the members should be notified again.

NOTICES

THE BOSTON CITY HOSPITAL

CONFERENCE OF CLINICAL PATHOLOGY

A monthly conference of clinical pathology will be held at the Boston City Hospital on Wednesday, March 10, at 12 o'clock noon, in the Pathological Amphitheater.

JOSEPH E. HALLISEY, M.D.,
Secretary, Medical Staff.

REMOVAL

CHARLES RICE, M.D., announces the removal of his residence from 14 Highland Street, West Concord, to 816 Blue Hill Avenue, Dorchester.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, March 11, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. Edward S. Emery, Jr., instructor in medicine, Harvard Medical School and associate in medicine, Peter Bent Brigham Hospital, will give a medical clinic. Practitioners and medical students are cordially invited to attend.

GREATER BOSTON MEDICAL SOCIETY

MEETING

Time: Tuesday, March 9, 8:30 p. m.

Place: Beth Israel Hospital Auditorium.

Speaker: Moses Behrend, M.D., surgeon to the Jewish and Mt. Sinai Hospitals, Philadelphia.

Subject: Carcinoma of the Colon. (Illustrated with moving pictures and lantern slides.)

Discussion: Carl Bearse, M.D., and R. B. Davidoff, M.D.

H. A. KONTOFF, M.D., *President*,
D. B. STEARNS, M.D., *Secretary*.

MASSACHUSETTS GENERAL HOSPITAL

A Clinical Meeting of the Staff of the Massachusetts General Hospital will be held in the Moseley Memorial Building, on Thursday, March 11, 1937, at 8:15 P.M.

PROGRAM

Case Presentations.

1. Recent Advances in Fever Therapy—Dr. Stafford L. Warren, Strong Memorial Hospital, Rochester, N. Y.

Physicians, medical students, nurses and social workers are cordially invited.

Committee on Hospital Meetings,
GRANTLEY W. TAYLOR, *Chairman*,
EARLE M. CHAPMAN, *Secretary*.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheater (Shattuck Street entrance), Tuesday evening, March 9, at 8:15 p. m.

PROGRAM

Presentation of Cases.

The Diagnostic Value and the Limitations of the Sternal Bone Marrow Biopsy. William Dameshek, M.D.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary*.

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the House of the Good Samaritan, Boston, Monday, March 8, at 8:15 p. m.

PROGRAM

1. Studies on the Use of Prontylin in Rheumatic Fever. Dr. Benedict F. Massell.
2. The Course of Rheumatic Fever and Heart Disease: A Fifteen Year Study. Dr. Edward F. Bland.
3. The Incidence of Recurrences of Rheumatic Fever. Dr. John P. Hubbard.
4. Clinical Analysis of Recurrences of Rheumatic Fever in Relation to Precipitating Events. Dr. T. Duckett Jones.
5. Hemolytic Streptococcus Studies in Rheumatic Fever. Dr. John R. Mote.
6. Short Summary of Virus Studies. Dr. T. Duckett Jones.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary*.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, March 16, at 12 noon.

The speaker will be Hyman Saphirstein, M.D., urologist to the out-patient department of the Beth Israel Hospital. His subject will be "Treatment of Gonorrhea."

All physicians are cordially invited to attend.

JOHN B. HALL, M.D., *Secretary*.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 8, 1937

Monday, March 8—

4 p. m. Boston Orthopedic Club. Warren Museum, Harvard Medical School.

5 p. m. Boston Orthopedic Club. Huntington Memorial Hospital.

6:30 p. m. Boston Orthopedic Club. Dinner, Vanderbilt Hall.

8 p. m. Boston Orthopedic Club. Amphitheater, Building D, Harvard Medical School.

*8:15 p. m. New England Heart Association. House of the Good Samaritan, Boston.

Tuesday, March 9—

*9 a. m - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Some Diagnostic Aspects of Neuroses Which Simulate Organic Disease. Dr. Felix Deutsch.

*8:15 p. m. Harvard Medical Society. Peter Bent Brigham Hospital Amphitheater (Shattuck Street entrance).

8:30 p. m. Greater Boston Medical Society. Beth Israel Hospital Auditorium.

Wednesday, March 10—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

12 m. Boston City Hospital, Conference of Clinical Pathology. Pathological Amphitheater.

†12 m. Clinicopathological Conference. Children's Hospital Amphitheater.

4 p. m. - 5 p. m. Surgical Pathological Conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

Thursday, March 11—

*8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Children's Hospital.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Cardiac Clinic. Dr. S. H. Proger.

*3:30 p. m. Medical Clinic at the Peter Bent Brigham Hospital. Dr. Edward S. Emery, Jr.

*8:15 p. m. Massachusetts General Hospital, Clinical Meeting of the Staff, Moseley Memorial Building.

Friday, March 12—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Some Observations on the Recent Outbreak of Epidemic Meningitis. Dr. Edwin H. Place.

*12 m. Boston Society for the Advancement of Gastroenterology. Carney Hospital, Boston.

Saturday, March 13—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

Sunday, March 14—

4 p. m. Free public lecture at the Harvard Medical School, Building D, Longwood Avenue, Boston. "Abnormal Terminations of Early Pregnancy." Dr. Arthur Hertig.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

March 4—Faulkner Hospital Clinical Meeting, 5 p. m.

March 5—William Harvey Society, 8 p. m., in the Auditorium of the Beth Israel Hospital, Boston.

March 6, June 8 and 9—American Board of Obstetrics and Gynecology. See page 78, issue of January 14.

March 8—New England Heart Association. See page 409.

March 8—Boston Orthopedic Club. See page 365, issue of February 25.

March 9—Harvard Medical Society. See page 409.

March 9—Greater Boston Medical Society. See page 409.

March 10—Boston City Hospital, Conference of Clinical Pathology. See page 408.

March 11—Medical Clinic at the Peter Bent Brigham Hospital. See page 408.

March 11—Massachusetts General Hospital. Clinical Meeting of the Staff. See page 409.

March 11—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

March 12—Boston Society for the Advancement of Gastroenterology, 12 noon, at the Carney Hospital, Boston.

March 16—South End Medical Club. See page 409.

March 29-31—First International Conference on Fever Therapy. See page 322, issue of February 18.

April 12-16—The Postgraduate Institute of the Philadelphia County Medical Society. See page 185, issue of January 28.

April 21-24—American Society for Experimental Pathology. See page 1075, issue of May 21.

July 6-11—Fifth International Congress of Hospitals. See page 37, issue of January 7.

October 5-8—American Public Health Association Meeting. New York City.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

April 7—Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. Francis B. Trudeau of Saranac Lake, New York. Subject: Personal Reminiscences of a Son of a Distinguished Father. (Illustrated.)

May 12—Annual Meeting. Salem Country Club. Speaker: Dr. P. P. Johnson of Beverly. Subject: A Trip to the Bahamas and Guatemala. (Illustrated.)

R. E. STONE, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of March and May.

CHARLES MOLINE, M.D., Secretary.

Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 16—Danvers State Hospital, Danvers.

May 11—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.

1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

March 30—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March 13—11 a. m Brockton Hospital.

April 15—Annual Meeting. 11 a. m. Ducy Hospital.

May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 31—Boston Medical Library. 8:15 p. m. "Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan. Discussion: Dr. Channing Frothingham.

April 28—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

March 10—The Memorial Hospital, Worcester. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14—Worcester Hahnemann Hospital, Worcester. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOK REVIEW

The Surgical Clinics of North America. Volume 16, Number 3, June, 1936. New York Number. 912 pp. Philadelphia and London: W. B. Saunders Company.

This issue of the Clinics consists of contributions by leading New York surgeons on a wide variety of major and minor conditions. A symposium on surgery for pain is headed by an excellent discussion of neurosurgical measures for the relief of pain presented by Byron Stookey. He outlines the nerve pathways for pain and then describes the various methods for temporary or permanent interruption of the pain impulses.

The common minor surgical problems that are discussed are athletic injuries to the muscles and tendons (G. G. Deaver), infections of the hand (Philip C. Potter), and injection treatment of hydrocele (Frederick W. Solley).

An interesting observation on the renewed interest in an almost discarded appliance is the fact that two different clinics recommend the use of the Murphy button in properly selected cases.

Several other clinics offering valuable information on topics of practical interest to the surgeon make this volume worthy of perusal.